DOCUMENT RESUME

ED 149 571 EC 104 165

AUTHOR Sigelman, C.; And Others
TITLE Human Rehabilitation Techniques. Disability Analyses: Motor Disabilities. Volume II, Part A.
INSTITUTION Texas Tech Univ., Lubbock.
SPONS AGENCY National Science Foundation, Washington, D.C. Office of Exploratory Research and Problem Assessment.
PUB DATE 77
GRANT ERP-75-10594; ERP-75-10594-A01
NOTE 221p.; A Technology Assessment; For related information, see EC 104 163-174

EDRS PRICE MF-$0.83 HC-$11.37 Plus Postage.
DESCRIPTORS Cerebral Palsy; Classification; *Definitions; Delivery Systems; *Demography; *Etiology; Incidence; *Individual Characteristics; Literature Reviews; *Physically Handicapped; Rehabilitation; *Services; Technology

ABSTRACT

Volume II, Section A of a six-volume final report (which covers the findings of a research project on policy and technology related to rehabilitation of disabled individuals) presents a review of literature on three types of motor disabilities--stroke, spinal cord injury, and cerebral palsy. Individual chapters on each disability cover the following: definitions and classifications; prevalence, incidence, mortality, and cost estimates; demographic distribution; etiology; life functioning deficits in the areas of health, motility, communication, cognitive-intellectual functioning, and social-attitudinal functioning; functioning as members of the community and labor force; technologies currently applied; and characteristics of the delivery system impacting the disability group. Bibliographies are also provided for each disability area. (SBH)

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HUMAN REHABILITATION TECHNIQUES

A Technology Assessment

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Volume II
Part A

Supplemental Report:
Disability Analyses
Motor Disabilities

EC104165

FOREWORD

This document is the second volume of the final report *Technology Assessment: Human Rehabilitation Techniques*, a project conducted at Texas Tech University by the Department of Industrial Engineering, the Research and Training Center in Mental Retardation, the Department of Systems, and the Technical and Professional Writing Program. The research has been conducted with the support of National Science Foundation grants ERP 75-10594 and ERP 75-10594 A01, monitored by the Directorate for Research Applications, Division of Exploratory Research and Systems Analysis.

Richard A. Dudek, Horn Professor and Chairman of the Department of Industrial Engineering has been a co-principal investigator and director of the project. Gerard J. Bensberg, Director of the Research and Training Center in Mental Retardation, and M. M. Ayoub, Professor of Industrial Engineering, have been co-principal investigators. Carol M. Sigelman and Andrew S. Martin of the Research and Training Center in Mental Retardation and Robert F. Powers of the Department of Industrial Engineering have been program managers for the project. James R. Burns and William M. Marcy of the Department of Systems have been in charge of the modeling used in the project. Technical writing has been performed by Charles W. Brewer and Cynthia E. Lyle of the Technical and Professional Writing Program. In addition, several research assistants and support personnel, as listed in the individual volumes of the study, have contributed to the project.

The project team wishes to acknowledge the efforts of individuals who have served on the Oversight Committee, Elizabeth Boggs, Kan Chen, Beatrix Cobb, Ronald Conley, Richard Herman, John Noble, Jr., Evan Vlachos, and Lester Wolcott; of Lee Phillips who served effectively as a program manager for a short time before leaving Texas Tech; of Brian Lambert who served as Work Session Conference Coordinator; of those who participated in the work-sessions chaired by Ted Hartman, David Malone, Blair Rowley, Evan Vlachos and John Wittman; and of Anne Seitz, the Secretary of the Project.

Although the National Science Foundation has supported this project, the findings, conclusions and recommendations expressed are those of the research team and do not necessarily reflect the views of NSF.

PREFACE

This volume is a supplement to the final report of the project "Technology Assessment: Human Rehabilitation Techniques." It includes reviews of the literature on 14 disabilities selected for intensive case study in the project. The contents of Volume 2 are as follows, with disabilities grouped on the basis of similarities of effects on functioning:

Section A--Motor Disabilities

Stroke	Cathy Mannion
Spinal Cord Injury	Linda Vengroff
Cerebral Palsy	Cynthia Spanhel, Linda Vengroff

Section B--Behavioral Disabilities

Epilepsy	Linda Vengroff, Melanie Schockett
Mental Retardation	Carol Sigelman, Linda Vengroff, Jerry Morris, Andrew Martin
Schizophrenia	Cynthia Spanhel, Melanie Schockett

Section C--Chronic Disease Disabilities

Rheumatoid Arthritis	Cathy Mannion, Cynthia Spanhel
Coronary Heart Disease	Cathy Mannion
Emphysema	Jody Dixon
Carcinoma of the Colon/Rectum	Linda Vengroff
Kidney Disease	Cathy Mannion, Bernadette O'Farrell Ray, Linda Vengroff
Diabetes Mellitus	Linda Vengroff

Section D--Sensory Disabilities

Visual Impairment	Melanie Schockett
Hearing Impairment	Melanie Schockett

These 14 disabilities were selected because they are prevalent, result in severe limitation, are chronic rather than acute, have survivors in need of rehabilitation, and affect a broad range of ages. The current literature on each disability was reviewed to determine: (1) definitions and classifications; (2) prevalence, incidence, mortality, and cost estimates; (3) demographic distribution; (4) etiology; (5) life functioning deficits in the areas of health, mobility, communication, cognitive-intellectual functioning, and social attitudinal functioning; (6) functioning as members of the community and labor force; (7) technologies currently applied; and (8) characteristics of the service delivery system impacting the disability group. The review papers vary in thoroughness as a function of the state of the literature about each disability and differences among the researchers doing the literature review. They reflect a first attempt at the ambitious undertaking of analyzing diverse handicapping conditions within the same analytical framework. The disability reports included in this volume provided the raw material for the cross-disability analysis reported in Working Paper 3, "Life Functions: Scope of the Problem of Disability," and in the final report Volume 1.

VOLUME 2

DISABILITY ANALYSES

VOLUME 2A - MOTOR DISABILITIES

VOLUME 2B - BEHAVIORAL DISABILITIES

VOLUME 2C - CHRONIC DISEASE DISABILITIES

VOLUME 2D - SENSES DISABILITIES

VOLUME 2A

MOTOR DISABILITIES

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CHAPTER 1
DISABILITY ANALYSIS: STROKE

I. Definition

In the literature on stroke, the following are the most common definitions:

Stroke (also called Apoplexy, Cerebrovascular Accident or Cerebral Vascular Accident) - an impeded blood supply to some part of the brain (American Heart Association, 1974 Heart Facts).

Stroke - a neurological deficit or cerebral dysfunction caused by a disturbance of the blood supply of the brain (Report of Task Force on Stroke Registries of the Joint Subcommittee on Cerebrovascular Disease of the National Advisory Heart Council and the National Advisory Neurological Diseases and Blindness Council, National Institute of Health, USDHEW, Sept. 15, 1967, p.10).

Stroke - an abruptly developing neurological abnormality which is secondary to an occlusive or hemorrhagic vascular accident involving one or more of the vessels (arteries or venous structures) of the brain (Hardin, et al., 1971, pp. 626-632).

Stroke - a sudden and severe seizure or fit of disease. The term is generally used for apoplexy and in connection with sunstroke and heatstroke.

In Apoplexy or "stroke" or cerebral hemorrhage, an artery in the brain either ruptures and bleeds or is blocked. The victim is apt to lose consciousness and some part of the body is paralyzed, at least temporarily (Fishbein, 1974, pp. 113, 1226-7).

Stroke - defined as the neurological result of an ischemic lesion in the cerebral hemisphere caused by arteriosclerotic or embolic occlusion with resulting hemiplegia (paralysis of one side) or hemiparesis (muscular weakness affecting one side of the body) (Stern, et al., 1971, p. 213).

Cerebral apoplexy (stroke) is understood to mean the acute occurrence of neurological symptoms caused by a vascular lesion in the brain, the brain stem

or the cerebellum. The two main groups are intracerebral hematoma and cerebral ischemic lesions (Paulson, 1971, pp. 327-360).

Apoplexy - "Apoplectic stroke; a condition caused by acute vascular lesions of the brain, such as hemorrhage, thrombosis or embolism, and marked by coma followed by paralyses. Also described as copious extravasation of blood within any organ (Extravasation - a discharge or escape, as of blood, from a vessel into the tissues) (Dorland's Illustrated Medical Dictionary, 1965, p. 120).

Cerebral vascular accident (CVA) - a stroke, an "accident" to or interference with the blood vessels that supply O_2 to the brain (The National Easter Seal Society for Crippled Children and Adults, Understanding Stroke, 1972).

Cerebral vascular accident - physicians use this term to refer to "stroke". A stroke occurs - usually suddenly - when an artery to a portion of the brain ruptures or is closed by thrombosis, embolism, or other causes.

Cerebrovascular or cerebral vascular disease (CVD) - diseases of the blood vessels serving the brain (National Heart and Lung Institute, and National Institute of Neurological Diseases and Stroke, 1972).

"Episode" ala Cerebral vascular accident - physicians usually speak of a CVA, meaning a stroke which has stricken someone else, but "your CVA" is a little too blunt, so when a physician must use the possessive pronoun, his standard reference is to "Your, uh, Episode" (Hodgins, 1971, p. 16).

Words used synonymously with stroke include apoplexy, cerebrovascular accident (CVA) cerebral embolism, cerebral hemorrhage, cerebral infarct, cerebral thrombosis, hemiparesis, hemiplegia, ictus, paralysis, paraplegia, shock, subarachnoid hemorrhage, subdural hematoma, transient ischemic attack or little stroke. Certain uncommon forms of stroke may bear the name of a physician such as Gerstmann's Syndrome, etc. (Licht, 1975, p. 1).

II. Societal Characteristics of Persons Affected by Stroke

The literature on stroke provides information about prevalence, incidence, mortality, and costs of the disability.

Prevalence

Approximately 2,00,000 persons now living in the United States have been victims of cerebrovascular disease at one time or another during their lives. This estimate encompasses major and minor involvement varying degrees of disability from practically none to severe incapacity. However, it is important to note that there are inherent flaws in Public Health Surveys from which these figures are derived. For example, minor episodes or unidentified attacks or symptoms are, in all probability, missed completely (Kuhn, 1963).

The American Heart Association (1969) released the following statistics on stroke: In 1965, nearly one-sixth of all patients admitted to some mental hospitals had CVD. In 1969 it was also reported that 1,620,000 living Americans had had strokes. Many of these patients had been rehabilitated and were leading productive lives. The 1971 statistics on stroke indicated that 1,650,000 Americans had been afflicted.

In the 1967 report of the Task Force on Stroke Registries of the Joint Subcommittee on Cerebrovascular Disease, prevalence information was extremely incomplete and neither the National Health Interview Survey nor the Health Examination Survey provided prevalence information on cerebrovascular disease.

Cerebral infarction associated with occlusive cerebrovascular atherothrombosis comprises more than 60% of all strokes (Heart Association Ed. Mat. No. 551). Embolic occlusion of the cerebral vessels constitutes the other major variety of occlusive cerebrovascular disease, accounting for 3-25% of all strokes (Mancall, 1975).

Incidence

Surveys indicate that the case rate for all strokes clearly identifies over 400,000 annual attacks in our present population (Kuhn, 1963; Horwitz, 1962). Incidence of stroke and its consequences may be expected to increase in the future with the increase of longevity (Zankel, 1971).

In the Framington, Mass. study, 5209 men and women classified while still asymptomatic according to their blood pressure, aged 30 to 62 were followed biennially over 14 years for the occurrence of strokes. 65 males and 70 females developed focal neurological deficits of abrupt onset meeting the minimum criteria for a stroke. 39 males and 47 females of these were diagnosed as atherothrombotic brain infarctions accounting for some 60% of all strokes (Kannel, et al., 1970).

In a retrospective study of all clinically distinguishable cerebrovascular disease syndromes admitted to Barnes Hospital (affiliated with Washington University School of Medicine, St. Louis) during a 1 year period, 130,500 patients were screened by clinics and emergency rooms, 32,077 patients were admitted as inpatients and CVD accounted for 490 (1.5%) of these admissions (485 separate patients) (Hardin, et al., 1971).

Since the latter part of the 19th century many articles have appeared on the natural history of cerebrovascular accident (CVA). The very wide range of statistics on this subject exists for several reasons:

1. The samplings of patients on which different reports have been based are extremely variable; some considered patients admitted to a general hospital or a general medical service, others based their reports on patients from neurological services. Still others obtained data from "chronic" wards, and several were based on the charts of patients in rehabilitation centers, either general or those specializing in neurological diseases.

2. Another factor which leads to a wide range of statistics is the arbitrary choice by authors of insufficiently precise terminology or the use of words which may mean different things to different workers. Some authors categorize patients into able-bodied, partially disabled, or disabled; others mention the degree of capability to perform activities of daily living; and still others speak of partial, incomplete or complete paralysis.

3. In discussions of symptoms, some authors mention headache or hypertonia instead of naming exactly what features were included in such terms, or speak of apnasia instead of specifiying type of aphasia, which makes the interpretation moot.

4. Frequently percentages are quoted instead of total numbers and are not broken down according to age, sex, etc., which would be required to validate any conclusions based on the number cited. This is especially important in relation to premorbid and postmorbid history.

Published data on strokes and the conclusions derived from them, are very much influenced by important mitigating factors. Facts and figures derived from public census tend to deal solely with clinically diagnosed cases and in these patients, the clinical diagnosis has not been substantiated by modern neurological testing techniques. Lack of this technical information is a critical flaw since lesions such as brain tumors, subdural hematomas and carotid artery obstructions, to mention but a few, may produce strokes which are externally identical. If a stroke clears up over a period of a few days, it often escapes notice; or conversely, if a patient dies suddenly of apoplexy, the death may be signed out as coronary occlusion without the benefit of autopsy. These factors lead to the omission of important data in public census (Kahn, 1963).

Mortality

The American Heart Association reports the 1971 statistics of death due to stroke as 207,920 (1974 Heart Facts). The 1967 report of the Stroke Registries reports the mortality from stroke as being high. Of those reported as experiencing a major stroke, approximately 50% die within one month and 66% die within six months.

Mortality changes in various disease categories are listed in Table 1-1. In most cases the total deaths increased despite declining mortality rates because of increases in the U.S. population. Since 1950, the death rate from stroke dropped 33% among middle-aged males (45-64 years) (American Heart Association, 1969).

TABLE 1-1

MORTALITY

Disease Category	1955-1965 Change in Mortality Rate Under 65	All Ages
	%	%
All Cardiovascular disease	- 7.1	- 5.1
Arteriosclerotic Heart Disease	+ 5.5	+11.1
Hypertensive Heart Disease	-46.1	-45.8
Cerebrovascular Disease	-19.7	-10.9
Rheumatic Heart Disease	-53.3	-32.8
Congenital Heart Disease		- 5.4
Other Cardiovascular Diseases		- 3.8

Source: National Heart Institute, 1969.

The overall reduction in stroke deaths may be a result of better care and treatment available in recent years, however the "reduction" may also in part be due to a change in coding introduced in 1959 by WHO, which has suggested that some of these deaths be labeled as the result of heart disease because of the concomitant arteriosclerosis and hypertension (Zankel, 1971).

According to Kuhn (1963) statistics maintained since 1900 show no

increase in the death rate from strokes despite the aging population. It is probable that these figures suffer from distortion introduced by increasing diagnostic discernment and that today's stroke figures are less likely to include patients with epilepsy, diabetic coma, uremia, etc.

The most serious factor in increasing the risk of stroke is high blood pressure. Many scientists attribute the nearly 20% reduction achieved since 1950 in mortality rates from stroke to the widespread application of effective drugs for the control of hypertension (National Heart & Lung Institute, 1948-73).

Kitowski (1970) reports that the National Office of Vital Statistics in 1964 indicated that diseases of the heart and blood vessels were the leading cause of death for all age groups. Deaths from specific diseases of the heart and blood vessels during that same year were estimated at 547,260 from CHD and 200,640 from stroke. Vascular diseases of the brain are now the third leading cause of death in the U.S. (Heart Association Ed. Mat. No. 551).

Age--Death rate from stroke is affected markedly with increased age. A sharp rise in deaths begins close to age 60 and 65% of fatalities from stroke occur at 65 or over (Kuhn, 1963).

Sex--Males die at a greater rate than females in almost every age group with the difference becoming striking from age 50 on. Recent studies indicate that this preponderance is linked directly to high incidence in the male of seriously diseased neck arteries (Kuhn, 1963).

According to the National Health Education Committee, Inc.(1971), in 1967, 16,042 more females than males died from a stroke. In figures this corresponds to 109,113 females and 93,071 males dying from vascular lesions affecting the central nervous system. Females in 1967 experienced a death rate that was 12% higher than the death rate for males from vascular lesions.

Zankel (1971) reports that stroke claims more female than male death victims in the U.S. (107,000 versus 94,000). Nonwhite females have the highest death rate from strokes, but the death rate for white females is lower than for white males.

Costs

In 1957, approximately 4,000 patients were discharged from New York City municipal hospitals with a diagnosis of some kind of stroke (Kuhn, 1963). The cost of one such patient who was hospitalized from January 8, 1960 to July 17, 1961 is as follows:

Doctor Bills Paid	$8,429.60
Hospital Bills Paid	7,496.00
Nurses Bills Paid	5,084.91
Cost of drugs prescribed	900.81
Gross	$21,911.32
Deduct: Covered by Insurance	7,891.31
Net cost to patient	$14,020.01

(Hodgins, 1971, p. 266).

Direct expenditure for services and supplies for stroke management amounted to $440 million in 1962. The total economic cost was stated at being $1.1 billion (Zankel, 1971).

The 1969 Public Health Service publication (No. 1921) "Patients in state and county mental hospitals 1967", indicated that 48,821 patients were in public, state, and county mental hospitals because of mental diseases due to cerebral arteriosclerosis and senile brain diseases (Fact Sheet Health Education Committee, 1971). These patients comprise approximately 15% of first admissions to public mental hospitals and about 13% of the resident population. As of 1971, when the committee published the fact sheet, 1967 figures were the latest such available information.

In an effort to estimate cost, the Health Education Committee used the above information and information contained in "Mental Health Statistics, Current Facility Reports, Provisional Patient Movement and Administrative Data, State and County Mental Hospitals U.S. July 1, 1967-June 30, 1968; and

July 1, 1968-June 30, 1969" compiled by the National Institute of Mental Health, Bethesda, Maryland.

The cost per year per patient under treatment in 1967 in state and county mental hospitals was estimated at $3,226. Thus the total cost of maintaining 48,821 patients in public mental hospitals with cerebral arteriosclerosis and senile brain disease in 1967 was $157,496,546. Yearly cost for patient care in 1969, assuming the same number of patients, was projected to be $224 million.

In fiscal year 1968, the National Heart Insitute (NHI) supported 48 research grants totaling $2,061,633 for studies on stroke. The following year grant money was allocated by NHI to the following studies.

Atherosclerosis & Coronary Heart Disease,	424 grants	totaling	$26,301,476
Hypertension ----------	269 grants	totaling	$12,388,570
Stroke ----------	36 grants	totaling	$ 1,218,823
Congenital Heart Disease----------	61 grants	totaling	$ 4,167,743
Rheumatic Fever & Rheumatic Heart Disease	40 grants	totaling	$ 2,093,809
Cardiopulmonary Disease----------	141 grants	totaling	$ 4,528,227
Heart Failure----------	530 grants	totaling	$29,251,578
Blood Components and Diseases----------	153 grants	totaling	$ 6,907,110
Other Cardiovascular Diseases----------	93 grants	totaling	$ 3,350,186
TOTAL	1,747 grants	totaling	$90,207,522

Source: National Heart Institute.

As can be seen from the above tables, stroke research accounted for approximately 2% of the number of funded grants and approximately 1% of the grant money allocated for cerebrovascular diseases in 1969.

The National Institute of Neurological Diseases and Stroke (NINDS) spent $5,772,000 for research in fiscal year 1969 (National Health Education Committee, 1971). In 1969, both NIH and NINDS spent an estimated $6,990,823 for research in cerebrovascular disease. Other agencies both private and public support cardiovascular research but is is impossible to determine how much is specifically spent in the area of cerebrovascular disease (strokes).

III. Demographics

Age--Almost all stroke victims are over age 40 and the majority are over 65 (Licht, 1975). Approximately 70% of strokes and 80% of stroke deaths occur among persons over age 65 and approximately 100,000 people experiencing their first stroke are under 65 (National Heart and Lung Institute, 1948-1973).

Although stroke is generally thought to be a disease of older persons, it strikes younger persons at an alarming rate. One in 6 of all stroke deaths occur at ages under 65 (American Heart Association, 1974). Stroke is the fourth leading cause of fatalities in the young productive age group (25-44) (Kuhn, 1965).

TABLE 1-2

AGE INCIDENCE OF VARIOUS TYPES OF CEREBRAL VASCULAR ACCIDENTS

Age	Hemorrhage (%)	Thrombosis (%)	Embolism (%)
Under 20	1	0	9
20 - 39	3	1	23
40 - 59	48	31	41
60 - 79	46	61	22
80 and over	2	7	5
	100%	100%	100%

Source: Hilleboe, 1955

In a study entitled Risk factors in stroke: a clinical study in Mexican patients, Olivares, et al. (1973) reported no sex difference in stroke frequency with 119 females (57.8%) and 87 males (42.2%) in the stroke sample. The ISSTE population at risk also included an excess of females in almost exactly the same proportion.

TABLE 1-2

SEX DISTRIBUTION - VARIOUS TYPES OF STROKE

	Thrombosis (%)	Hemorrhage (%)	Embolus (%)	Total
Female patients	54	61	68	57

Source: Olivares, et al. (1973).

Ethnicity--Nonwhite persons die of stroke at a greater rate, 2 to 6 times greater than that of white persons. The differential increases with adjustment for age, indicating that nonwhite death rates are much higher than white rates even in younger age groups. For each 100,000 living in the 40-44 age group, 75 nonwhite are killed by stroke, compared to 15 white persons. A partial explanation of this difference is the tendency to call any acute medical calamity a stroke when it occurs to a nonwhite, but it is also likely that intracranial aneurysms, for example, occur with somewhat greater incidence in the Negro race (Kuhn, 1963).

The American Heart Association (1974) profiles a candidate for stroke as one with high blood pressure and a history of brief, intermittent stroke episodes. Examinations often show evidence of hardening of the arteries (atherosclerosis) in the heart, neck and legs and indications of diabetes. Tests may reveal increased cholesterol level and other fats in the blood. Black Americans are twice as likely to have high blood pressure as whites and as the above profile suggests, this makes blacks more prone to stroke and heart attacks. Blacks suffer more strokes at an earlier age and with more severe results. The reasons for

this is not fully understood. Perhaps heredity, life style, diet and stress play a role in this (American Heart Association, 1974).

Socioeconomic status or education--Insofar as is known, stroke is no respector of occupations; as a matter of fact, individuals who are generally prone to develop coronary insufficiency seem also susceptible to cerebral vascular insufficiency. Cerebral ischemia is produced by atherosclerotic lesions in the sedentary American male laboring under high tension in a manner similar to production of cardiac ischemia. Heart failure and brain failure are linked by the common denominator, atherosclerosis (Kuhn, 1963).

In a report on social disorganization and stroke mortality in the black population of North Carolina, Neser, et al.(1971) reported that the mortality rate due to stroke rose steadily as indices of family disorganization increased. The largest death rate differential across county groups of social disorganization was 2.5 times in the 35-44 age group. This relationship remained but decreased in magnitude as age increased. The authors believe that it is unlikely that this differential is a function of income level or geographic location, and the findings may reflect a physiological response to the constraints imposed upon subordinated members of society. This has been confirmed in animal studies where the effects of constraints upon subordinated members of a group bring about endocrine changes and increases susceptibility to disease.

Urban-rural--In a poll on medical practice conducted by Modern Medicine, it was found that the incidence of strokes due to thrombosis was lower (56%) in communities under 5,000 population than in larger cities. Corresponding figures were 66% in cities of more than 500,000 people, 68% in the population group of 50,000 to 500,000, and 60% in places of 5,000 to 50,000 inhabitants.

Geographic region--The President's Committee on Heart Disease, Cancer and Stroke (1964-'65) found a definite geographical pattern for stroke death rates, with the highest death rates occurring in the Southeastern states and the lowest in the southwestern and mountain regions (Zankel, 1971).

In some Southern states, strokes are said to constitute the second leading cause of death (a conclusion which may represent a real difference in the races or the use of the word "stroke" may be used to describe almost any sudden death) (Kuhn, 1963).

IV. Etiology

The three main causes of cerebral vascular accidents (CVA) are cerebral thrombosis, cerebral hemorrhage and cerebral embolism. Compression and spasm are sometimes included as two additional ways cerebrovascular disease (CVD) may occur (National Heart and Lung Institute and National Institute of Neurological Disease and Stroke, 1969).

A poll by more than 6,800 readers of Modern Medicine, found that of the 12,390 strokes reported, 7,983 (64%) were diagnosed as having been caused by thrombosis, 2,336 (19%) were considered to have been caused by hemorrhage and 897 (7%) were due to embolism, with the remaining 10% happening because of other or unknown causes (1969).

Thrombosis (clot formation). "A clot of blood may form in an artery of the brain and may stop the flow of blood to the part of the brain supplied by the clot-plugged artery" (National Heart & Lung Institute & National Institute of Neurological Diseases & Stroke, 1969).

According to the American Heart Association (1969), cerebral thrombosis is one of the most common causes of strokes. Cerebral damage is slight if an arteriole (a minute arterial branch, especially one just proximal to a capillary) is involved, but serious effects result when a major artery supplying blood to the brain is affected by the clot and no collateral circulation is present (Zankel, 1971).

Thrombosis of a cerebral artery most often is preceded by atherosclerotic changes in the vessel (Hilleboe, 1955). The abnormal condition of atherosclerosis is when thick, rough deposit forms on the inner wall of the artery. This build-up reduces the blood flow and decreases the natural elasticity of the vessels. Blood clots often form

in such diseased arteries and, depending on the size of the clot, may shut off the passageway of a cerebral artery (American Heart Association, 1969; Zankel, 1971).

Cerebral thrombosis may also form in the presence of inflammation of the cerebral vessels. This inflammation may be primary as in periarteritis nodosa (an inflammatory disease of the coats of the small and medium sized arteries of the body with inflammatory changes around the vessels and marked by symptoms of systemic infection) or may be secondary to acute meningitis or to acute encephalitis.

Hemorrhage (bleeding). "The wall of an artery of the brain may break, permitting blood to escape and thus damage the surrounding brain tissue" (National Heart & Lung & National Institute of Neurological Disease & Stroke, 1969).

Of the various types of CVA's, cerebral hemmorrhage causes the most extensive damage to the brain tissues (Hilleboe, 1955). Since hemorrhage into the brain substance takes place when the artery wall cannot withstand the pressure of the blood within it, the event is often associated with moderate to severe hypertension (Millikan, 1963). In addition to hypertension, strokes caused by hemorrhage may result from congenital anormalitities of the cerebral vessels, deep-seated aneurysms, some tumors, and from trauma or head injury.

Embolism (Blocking of a vessel by a clot floating in the blood stream) "A clot from a diseased heart or, less commonly, from elsewhere in the body may be pumped to the brain and stop up one of the brain's arteries" (National Heart & Lung Institute & Institute of Neurological Disease & Stroke, 1969).

The type of embolus (clot, fibrin platelet, cholesterol, fat, air, tumor, metastasis, bacterial, foreign bodies) varies with the patient's age according to Toole and Patel (1974). In young adults, emboli subsequent to rheumatic valvular heart disease tend to occur, while in older patients the most often observed are emboli secondary to cholesterol plaques. While a blood clot originating from the heart as a result of valvular or endocardial disease is the most frequent cause of cerebral embolism, other embolism sources are paradoxical (occurring at variance with the normal rule), lungs, pulmonary veins, and aortocranial arteries. Prosthetic (artificial) valves (Medical World News, 1971), atrial fibrillation, myocardial infarction, cardiac surgery, angiography, (Millikan, 1973), trauma due to fat embolism which occasionally complicates trauma to long bones, or to air embolism developed by too rapid changes in atmospheric pressure resulting in Caisson Disease ("the bends"), various parasites and ova which lodge and encyst in the small arteries of the brain, carcinomas of the lung, breast, stomach, kidney, thyroid, as well as malignant tumors which migrate to and lodge in the brain where they enlarge and act as tumors (Toole & Patel, 1974) are additional causes of embolism.

Compression "A tumor, swollen brain tissue, or a large clot from another vessel may press upon a vessel of the brain and stop its flow of blood" (National Heart & Lung Institute and National Institute of Neurological Disease & Stroke, 1969).

Spasm (tightening and closing down of the walls of an artery) "An artery of the brain may constrict and thus reduce the flow of blood to an area of the brain."

If the spasm is of short duration, permanent damage does not necessarily result (National Heart & Lung Institute and National Institute of Neurological Disease & Stroke, 1969).

While the walls of the smaller cerebral arterioles do not appear to have the muscle or elastic tissue necessary for construction, the larger extracranial arteries do have elastic and muscle fibers which allows these arteries to experience spasms. Hypotension or anemia when associated with such narrowing of the artery favors cerebral infarction (in which an area of the brain is deprived of blood supply producing a neurological deficit in affected area) (Zankel, 1971).

Occurrence in the young. Rupture of a congenital defect in a blood vessel is the most frequent cause of stroke in the young (National Heart & Lung Institute and National Institute of Neurological Disease & Stroke, 1969).

Some studies have investigated the possible relationship between "the pill" and stroke. The evidence when compared to the large number of females taking the drugs, at best seems to support that the use of oral contraceptives is associated with an increased risk of thrombotic stroke. Therefore "the pill" should not be prescribed for females who have hypertension, severe migraine, or migraine associated with focal neurological signs, history of stroke, venous thromboembolism or depression (Fryer, Tytus, & Pardee; Heyman & Hurtig, 1975).

Risk factors. Hypertension, overweight, cigarette smoking, hypercholesteremia, atherosclerosis, heart disease, diabetes mellitus and TIA's are all considered by National Heart & Lung Institute and National Institute of Neurological Disease & Stroke, (1969) to be risk factors for stroke.

Hypertension--Atrial hypertension is recognized as the commonest and greatest risk factor to the development of thrombotic and hemorrhagic strokes (Lavy, Melamed, Cahane & Carmon, 1973; #21). While the basic cause of most high blood pressure has not been identified, there are a wide variety of drugs with which to control it (American Heart Association, 1974). Early detection of hypertension by a doctor and subsequent treatment can help reduce stroke risk, as there is a latent period in hypertensive disease before its cerebral sequelae appears (Kannel, Wolf, Verter & McNamara, 1970).

In the study reported by Lavy, Melamed, Cahane and Carmon (1973), 42% of the total stroke series had hypertension, with the maximum frequency of hypertension among stroke cases in the 60-69 years of age group.

Obesity--People who are obese can be expected to eat diets high in sugar and saturated fats. Most likely, these individuals are prone to high blood pressure, high cholesterol and atheroselerosis which as mentioned are risk factors for stroke. Additionally, obese people rarely enjoy sufficient exercise.

Cigarette smoking--Risk of a stroke increases in direct relationship to the number of cigarettes smoked (American Heart Association, 1974).

Hypercholesterolemia (excess of cholesterol in the blood)--Hypercholesterolimia increases the risk of cerebral thrombosis. Fryer, Tytus, and Pardee state that it is not known whether reducing cholesterol levels by diet or drugs reduces the incidence of stroke.

Atherosclerosis--The underlying cause of most strokes in atherosclerosis affecting the blood vessels of the brain itself or the brain's major blood

supply arteries in the chest and neck (25th Anniversary of Heart & Lung Institute). For a more complete description of the process refer to the previous section Thrombosis in this report. In the present series of disability reports for the technology assessment grant, factors such as diet, lack of exercise, etc., which promote atherosclerosis are discussed in the Coronary Heart Disease Report.

Heart disease--A high incidence of heart distrubances is characteristic of stroke patients (refer to Embolism). Kuhn (1963) reports that heart failure has been found concomitantly in almost half of the patients dying of cerebral infarction.

Diabetes mellitus--Diabetes, or a familial tendency for this disease, is associated with increased risk of stroke (American Heart Association, 1974). The disease contributes to atherogenesis (formation of atheromatous lesions in artereal walls) in the cerebral vessels by the multiple lipid disorders associated with diabetes

Transient stroke or transient ischemic attack (TIA)--If stroke episode effects clear within a few minutes to a few hours, the person is considered to have experienced a TIA. A CVA lasting over 12 hours is considered a stroke (Baker, 1963). If the cerebral blood supply is interrupted for a few hours, it is referred to as an advancing stroke or a stroke in evolution. Permanent interruption of the blood supply indicates a complete stroke (Zankel, 1971).

Ostfeld, Shekelle and Klawans (1973) in a prospective study of 2,772 poor people aged 65 to 74 years observed that white males, black males and black females with TIA had a higher rate of observed stroke than persons without TIA. White females were a notable exception.

Hereditary There is no evidence that stroke or the contributing atherosclerotic process are hereditary, but a tendency for these disorders can be inherited. The incidence of such problems is higher in some families and familial susceptibility is identifiable (American Heart Association, 1974).

Cultural A study of 206 clinically diagnosed Mexican stroke patients who were hospitalized supported the identification of risk factors that were previously reported as important in mainly northern populations. From the data, it was concluded that ethnic and cultural variability exerted little effect on the likelihood of experiencing a stroke (Olivares, Castaneda, Grife, Alter, 1973).

Season of the year and weather The study by Olivares, et. al., (1973) showed a sharp increase for cerebral thrombosis in August and September which corresponds to the end of the Mexican rainy season. These two months also showed an increase for cerebral hemorrhage. Cerebral embolism was above the mean in March and May, and August and September.

Miscellaneous Other factors such as occupation, occurrence of stroke incidence, in relation to holidays, time of day and weather were also investigated in the Mexican study by Olivares, et. al., (1973) but are not reported since no corresponding information was found for American conditions.

TABLE 1-4

LIFE FUNCTION TABLE - STROKE: MOBILITY

Problem	Dynamics	Source
Paralysis	Extent can only be established after stroke patient has regained consciousness. When due to temporary condition such as pressure on a nerve rather than to actual destruction of nerve tissue will disappear.	M. Fishbein, 1974.
Trunk Muscle Paralysis (including abdominal muscles)	Weakness of the trunk muscles will tend to cause the patient to lean to the paretic side because the erector spinea is unable to keep the spine upright. Interferes with proper sitting, standing, walking.	H. T. Zankel, 1971.
Spasticity (Dyskinesia)	Prevents patient from putting foot on ground-causes him to draw his foot and walk on his toes.	
Clonus-spasm in which rigidity and Relaxation alternate in rapid succession	Frequently manifested by marked clonus-prevents ambulation altogether.	
Dysequilibrium-when equilibritory muscular activity is disturbed	Stroke patient, during attempted walking or standing	H. T. Zankel, 1971; Understanding stroke, 1972; H.E. Hilleboe 1955.
Persistence of flaccid paralysis	Hemiplegia-usually only during initial period of unconsciousness. Small projection when lasts longer than 48 hours, may persist for weeks or months	H. E. Hilleboe, 1955; E.E. Gordon, 1963.

TABLE 1-4, CONT'D.

Problem	Dynamics	Source
Floor and ground surfaces-Elevation activities must learn to stand up, sit down, climb stairs and curbs, cross the street	Hemiplegic patient	H. E. Hilleboe, 1955;
Akenetic mutism-state of unresponsiveness to the environment with extreme reluctance to perform even elementary motor activitives		J. M. Segarra and N. Angelo, 1970.
Hemiplegia Loss of voluntary motor power but no loss of reflex activity	National Health Survey-200,000 cases complete paralysis and 100,000 with partial or slight paralysis. Spontaneous improvement up to 6 months. Most frequent findings in stroke reported motor deficit-92% of patients. Upper limb always involved, lower limb affected 83%	R.A. Kaelin, 1963; H.E. Hilleboe, 1955 H.T. Zankel, 1971; J.P. Held, 1975; S. Brunnstrom, 1970.
Stiff or weakened joints	Can be prevented or lessened by nursing and exercises.	Understanding stroke, 1972.
Reduced mobility and leg strength	Practically no improvement observed 2 months after stroke. Improvement on involved side generally small and usually without clinical significance.	P.H. Stern, F. McDowell, et al., 1971.
Shortened heel cord with plantar flexion of the foot	Common complication of hemiplegia must be corrected if patient is ever to walk again.	H. E. Hilleboe, 1955;

TABLE 1-4, CONT'D.

Problem	Dynamics	Source
Flexion contractures of the knee	Stroke patient	H.E. Hilleboe, 1955;
Ambulation	If shortly after CVA, there is any residual movement in the hemiplegic's paralyzed arm, probable that spontaneous return of function to the leg will suffic to permit ambulation. Most stroke patients able to return home, ambulate, become independent, at least partially. Registry statistics: 518 patients, 85% became ambulatory; only 47% ambulation in sensory-involved patients; Registry 124 patients, 61% ambulatory independently with or without mechanical aids.	H.E. Hilleboe, 1955; M. Di Benedetto, 1974;
Atrophy of sound arm and leg	If left too long in bed before rehabilitation begins.	H.E. Hilleboe, 1975;
Bed activities change position, move around, sit up, get out of bed.	Hemiplegic patient must be taught. More readily performed if patient sleeps with paralyzed side away from the side of the bed from which he gets up	H.E. Hilleboe, 1955;
Wheelchair activities, get in and out, propel it.	Hemiplegic patient must be taught to use good arm and leg to best advantage.	H.E. Hilleboe, 1955;
Bed transfer	Left hempiplegics tend to do less well on this task than right hemiplegics.	L. Diller, 1963;
Gait problems	Hemiplegia	S. Brunnstrom, 1976;

TABLE 1-4, CONT'D.

Problems	Dynamics	Source
Motor behavior problems. Basic limb synergies, attitudinal or postural reflexes, associated reactions, hand reactions.	Adult patients with hemiplegia	S. Brunnstrom, 1970;
Dyskenesia	Loss of motor power, associated spasticity and sometimes incoordination.	H.T. Zankel, 1971;
Public conveyance. main problem lies in negotiating the first long step up from street level	Hemiplegic patient	H.E. Hilleboe, 1955;
Driving-personal automobiles	Difficult for the hemiplegic unless has some degree of usefulness in involved hand and arm. Best interests of safety-most should be dissuaded from trying to drive. Refer to state motor vehicle laws.	H.E. Hilleboe, 1955; Lombardi,
Falls	Hemiplegics tend to fall more frequently than other persons. Most common during rising from a sitting to standing position, when patient tries to walk in poorly lighted room, when others pass too close to patient's uninvolved side, when patient allows good side to come too close to objects (turning a corner or passing through a door)	H.E. Hilleboe, 1955;

TABLE 1-4, CONT'D.

Problems	Dynamics	Source
Impared motor function of upper extremity	Study cited-116 hemiplegic patients-40% who recovered function completely had initial motion within the first month after stroke full motion occurring most often in first month and within 3 months, 40% partial recovery-process was maximized by the 6th-7th months.	J.E. Sarno, Jr., 1967;
Loss of mobility, loss of muscular strength and endurance	Deteriorations that can occur in patients confined to bed-Secondary effects	Notes taken by a physician
Hand function	Relatively few persons having completed strokes regain useful hand function.	E.E. Gordon, 1963;
Adiadochokinesis or dysdiadochokinesis inability to execute alternating and successive movements rapidly and smothly smoothly	(Trouble playing pat-a-cake) stroke victim	H.T. Zankel, 1971;
Incoordination or dyssynergy-overall term for disturbed motion	Stroke patient homolateral atonia (deficient muscular tonicity), disorders of muscular contraction, disorders of voluntary movement, tremor	H.T. Zankel, 1971;
Intricate activities that require use of both hands Playing the piano, violin. Inability to perform fine finger skills. (buttoning, knitting, sewing)	Most cases will be denied hemiplegic	H. E. Hilleboe, 1955;

TABLE 1-5

LIFE FUNCTION TABLE - STROKE: HEALTH

Problem	Dynamics	Source
Severe pain in exremity resulting from inability to change position. Sitting may become unbearable.	Leg so weak if not propped and positioned will rotate outward feeling in time as if enormous weight were pulling it from socket.	F. McNeel, 1975;
Ischemic ulcers, circulatory deterioration, metabolic imbalance, urinary tract dysfunction, respiratory complications.	Deteriorations that can occur in patients confined to bed-Secondary effects	Notes taken by a physician
Unconsciousness, labored breathing	May cause death	L. Galton, 1975;
Self-Care activities	Hemiplegic. Can be taught to do most with one hand	H.E. Hilleboe, 1955;
Severe headaches	May be experienced by some aphasic patients	Aphasia and the family, 1965;
Convulsions (seizures)	Some aphasic patients may be subject to such experiences	Aphasia and the family, 1965;
Fatigue	Most brain damaged patients have unpredictable periods of extreme neurological and physical fatigue	McKenzie Buck, 1963; Understanding stroke, 1972; F. McNeel, 1975.
Permanent deformities. Fist closed over thumb, foot-drop, frozen shoulder, external rotation of the hip, hyperflexion of the knee.	Sometimes flexion of fingers over thumb so severe that nails dig into palm. Some deformities can be prevented by good bed care.	C. Mattingly, 1963; H.E. Hilleboe, 1955; H.T. Zankel, 1971;

Problem	Dynamics	Source
Contracture to weight-bearing joints, hip, knee (condition of fixed high resistance to passive stretch of a muscle, resulting from fibrosis of the tissues supporting the muscles of the joints, or from disorders of muscle fibers)		Notes taken by a physician
Pressure cone at the foramen magnum (foramen magnum-the large opening in the anterior and inferior part of the occipital bone, interconnecting the vertebral canal and the cranial cavity)	Can develop as a result of cerebral edema if head is elevated above pillow height when the patient is unconscious	C. Mattingly, 1963;
Sensory perceptual defects-faulty perception of verticality -loss of position sense	Hemiplegic patients seem to err more especially in the vertical position.	E. E. Gordon, 1963; L. Diller, 1963;
Astereognosis-inability to recognize objects or to appreciate their form by touching or feeling them.	Extent of error related to ability in learning to ambulate. Right and left hemiplegics display systematic errors	
Somatic (pertaining to the body) or spatial delusions or hallucinations	Common consequences of focal cerebral lesions when sensory or perceptual functions disturbed	S. Horenstein, 1976; F. McNeel, 1975;
Involuntary muscle contractions in the affected upper limb	Frequently accompanies a yawn in hemiplegic patient.	S. Brunnstrom, 1975;

TABLE 1-5,CONT'D.

Problem	Dynamics	Source
Muscular contractions	coughing and sneezing in hemiplegics can evoke sudden muscular contractions of short duration	S. Brunnstrom, 1970;
Sacral pressure sore (ulcer)	Permanent. Compromises the amount of sitting that the patient will be able to do in the future. Preventable.	Notes taken by a physician
Edema	Of the hand. Preventable by supporting extremity	Guide lines to stroke patient care 1975; E. E. Gordon, 1963;
Sensation deficits diminished or destroyed sensations of touch, pain, pressure, heat and cold. See pallhypesthesia, pallanesthesia	Usually loss of or reduced sensation on same side of paresis-opposite locale of CVH,partial loss of sensation on the homolateral side	Guidelines to stroke patient care, 1975; McKenzie Buck, 1957; H.T. Zankel, 1971;
Diplopia-perception of 2 images of a single object	Slowly improves-6 months for eyesight to approach normal	F. McNeil, 1975;
Incontinenee of urine (persistant urinary inconinence beyond 6 months in 20% of patients	Hemiplegic patient-result of brain damage from CVA when there is extensive encephalomalacia (softening of brain). Usually product of prolonged bedrest and lack of motivation. Bladder control in these cases usually returns when patients gotten out of bed and started on active rehabilitation program	H. E. Hilleboe, 1955; M. DiBenedetto, 1974.
Paralysis of lower 2/3's of one side of face	Not uncommon in hemiplegia. Most often on the affected side. Function usually returns.	H. E. Hilleboe, 1955;

TABLE 1-5,CONT'D.

Problem	Dynamics	Source
Nasopalatal paralysis-manifested by weakness of phonation and shallow respiration	Occasionally seen as a stroke symptom due to the involvement of vertebrobasilar system. Usually associated with paresis of the intercostal muscles	H. T. Zankel, 1971;
Paralysis of the tongue	If patient has a sharp or broken tooth-tongue can be scratched, choke if tongue falls to back of throat.	H.T. Zankel, 1971; C. Mattingly, 1963;
Paralysis of the lips		H. T. Zankel, 1971;
Thalamic crying-patient will cry without tears at the slightest provocation	Well recognized manifestation of dysmentia. Uncontrollable	H. T. Zankel, 1971;
Loud rhythmic laughing	Manifestation of dysmentia. Uncontrolable	H. T. Zankel, 1971;
Thalamic pain-Burning pain in paretic side. Usually worse in extremities* particularly in the hand	Several weeks after onset of stroke May persist for months.	H. T. Zankel, 1971
Pallhypesthesia-diminished sensation for vibration	Dysesthesia	H. T. Zankel, 1971
Pallanesthesia-complete loss of sensation in the paretic extremities and some loss in the monparotic extremities	Dysesthesia	H. T. Zankel, 1971;

Problem	Dynamics	Source
Hygensia-loss of sensation of taste	Dysesthesia-not common in stroke patients	H. T. Zankel, 1971;
Hypogensia-diminished sensation of taste	Dysesthesia	H. T. Zankel, 1971;
Parageusia-hallucinations of	Dysesthesia	H. T. Zankel, 1971;
Anosmia-loss of sense of smell	Dysesthesia	H. T. Zankel, 1971;
Parosmia-hallucinations of smell	Dysesthesia	H.T. Zankel, 1971:
Bilateral homonomous hemianopsia-causes blindness in ½ of each eye	Dysesthesia. Common symptom following a stroke	H. T. Zankel, 1971 F. McNiel, 1975;
Extinction-inability to appreciate double simultaneous stimuli	Dysethesia. Many patients suffer from extinction	H. T. Zankel, 1971;
Decubiti-ulcer	Due to recumbent (lying down) position	C. Mattingly, 1963;
Hypostatic pneumonia	Turning at frequent intervals can help prevent	C. Mattingly, 1963:
Venous thrombosis	Turning at frequent intervals can prevent	C. Mattingly, 1963;

Problem	Dynamics	Source
Eating and swallowing	If hemiplegic patient does not concentrate-will choke. Food must remain on unaffected side to go down safely	F. McNiel, 1975; R. N. Baker, 1963;
Loss of eyeblink reflex-if undetected can develop ulcerated cornea		E. Hodgins, 1971;
Drooling	Patients with heavy cranial nerve involvement	E. Hodgins, 1971;
Accumulation of large amounts of food on affected side of mouth	Patients with heavy cranial nerve involvement	E. Hodgins, 1971; H. T. Zankel, 1971;
Glove and stocking effect of affected side	Cold temperature intensifies pins and needles feeling. Jar of affected heel on sidewalk makes sensation rise almost to threshold of pain	E. Hodgins, 1971;
Shoulder pain-radiates down the arm and hand	Frequently found in patients with hemiplegia. Several weeks after stroke.	V. J. Kitowske, 1970, H. T. Zankel, 1971,

LIFE FUNCTION TABLE - STROKE: SOCIAL-ATTITUDINAL

Problems	Dynamics	Source
loss of pleasure derived from food	Diet-low fat, low salt, low cholesterol and childlike foods. Discouragement and loss of appetite because of having to be fed by someone else	F. A. Whitehouse, 1963; F. McNeil, 1975;
Wernicke's aphasia (cortical sensory aphasia) can present the most dramatic psychiatric syndrome	Hemiplegias usually aren't present. Patient generally appears quite unaware of disturbance-frequently euphoric	N. Geschwand, 1970;
Broca's aphasia- (ataxic aphasia, lacking coordination) depressed, tearful, weeps at assurance, distressed by errors in testing	loss of visumotor skills in stroke patient	N. Geschwend, 1970;
Mechanisms of belittling, minimizing or pushing away reminder of the disability	Patient characteristically responds to the sudden onset of his disability in a way that appears appropriate	L. Diller, 1963;
Neurotic or psychotic reactions of any kind	Depends on premorbid personality	A. Shulman, 1971;
Projection	May be used when high level, well-organized defensive systems breakdown entirely or alternatively	H. Shulman, 1971;
Anxiety	Experienced as defensive operations weaken in a stroke patient. Loss of verbal skills. Intensity can vary from a vague sense of unease to frantic attacks of panic.	H. Shulman, 1971; L. Diller, 1963;

Problem	Dynamics	Source
Easily emotionally aroused-may show anger suddenly feel depressed, laugh or cry easily or unexptectedly	After stroke less control over feeling, than before illness	Understanding stroke, 1972.
Eating-frightening chore	Trigeminal (5th craneal nerve) involved. Half of mouth paralyzed, hands paralyzed so finger can't retrieve food.	F. McNiel, 1975;
Lapses in tact and social sensitivity Coarsening of manners and morals	subtle alterations-somtimes observed-often largely observed by enourmous physical deficits	H. Shulman, 1971;
loss of consideration		H. Shulman, 1971
Irritability		H. Shulman, 1971
Regression	it is a result of the stroke experience	H. Shulman, 1971;
Denial-patient can't accept disability and has to acquire compensatory skills	holds on to illusion that complete cure will come with additional treatment. Can't be discouraged	H. Shulman, 1971;
Paranoid psychotic disorders-delusional states of jealousy	Not uncommon. Occasionally accompanied by murderous attacks of the spouse or suicide	H. Shulman, 1971;
Loss of worth	Disabled stroke patient	Understanding stroke, 1972;
Childish dependency	Childlike foods support this feeling	F. A. Whitehouse, 1963;

Problem	Dynamics	Source
Stroke experience reminder of mortality	Stroke is always a shock. Consciously or otherwise regarded as a step closer to death	F.A. Whitehouse, 1963;
Stroke-conviction of sin	commonly perceived interpretation of the stroke experience	W. B. Daylong, 1974;
Anger	Emotional reaction to deficits	W. B. Daylong, 1974;
Feelings of isolation and loneliness	emotional reactions to stroke experience and deficits	W. B. Daylong, 1974; F. A. Whitehouse, 1963.
Reluctance to discontinue completed formal rehabilitation program	Believe normal function will be regained if treated long enough	J. E. Sarno, Jr., 1967;
Preoccupation with fantasy		F. A. Whitehouse, 1963;
Systematized referential or paranoid thinking	Common consequences of focal cerebral lesions when sensory or perceptual functions disturbed	S. Horenstein, 1970;
Autism-condition of being dominated by subjective, self-centered rends of thought or behavior	Common consequences of focal cerebral lesions when sensory or perceptual functions disturbed	S. Horenstein, 1970;
Paranoid responses		M. Ullman, 1963;

Problem	Dynamics	Source
Anosognosia-loss of ability in a person to recognize that he has a disease or a bodily defect		M. Ullman, 1963;
Negative and morbid feelings resulting from severe blow to ego and morale	Reaction to stroke attack suddenly causing at least partial paralysis, some brain damage & mental confusion	F. McNeil, 1975;
Confusion	Patient begins to comprehend neurological damage. Mild except when overtired.	F. McNeil, 1975;
Concern for acceptance by others	Develops after patients initial confusion about intellectual loss	F. McNeil, 1975;
Anguish-over common visual hallucinations	Builds from repression that such experiences mean patient is a psychiatric case	F. McNeil, 1975;
Depression, mania, overt schizophrenic disorganization-reactions of frank psychiatric illness	Don't occur with any consistent degree of regularity	H. Shulman, 1971;
Depressive reaction-mild& transient states of unhappiness to full blown neurotic or psychotic states	Need to ventilate the severe psychological trauma, energies of intellectual as well as physiological areas affected. Contributed to by inability to do simple tests.	McKenzie Buck, 1957; M. Ullman, 1963; E. Hodgins, 1971; H. Shulman, 1971; S. Horenstein, 1970.

TABLE 1-7

LIFE FUNCTION TABLE - STROKE: COGNITIVE-INTELLECTUAL

Problem	Dynamics	Source
Failure in judgment and planning	Subtle alterations-sometimes observed-often largely obscurred by enourmous physical deficits.	H. Shulman, 1971;
Decrease in powers of grasp and comprehension		H. Shulman, 1971;
Organicity-refers to underlying perceptual disturbance brought about by brain damage	Roughly ½ the hemiplegics treated in a rehab center show signs of organicity ½ do not.	L. Diller, 1963;
Limited memory span-may not remember well or pay attention very long		Understanding stroke, 1972;
Dysmentia - the neurotic and/or psychotic manifestations in stroke patients as part of the stroke syndrome	Usual findings-confusion of the mind, inability to think, inability to remember	H. T. Zankel, 1971;
Impersistence-inability to concentrate upon object or examiner-failure to remember to put out lighted match	Commonly found in stroke patients	H. T. Zankel, 1971;
Perseveration-counting stop at number, repeat, go on-try to turn on gas after turning it on	Manifestation of dysmentia	H. T. Zankel, 1971;
Blotting out of area of memory center	stroke aftermath	L. Galton, 1975;

TABLE 1-8

LIFE FUNCTION TABLE - STROKE: COMMUNICATION

Problem	Dynamics	Source
Paralysis-inability to communicate needs. If hospital call button not placed securely in hand, no way to get needed attention	Patient may be reported incontinent when real problem is inability to ask for bed pan.	F. McNeil, 1975;
Defective verbal skills	In a learning situation may impair performance	L. Diller, 1963
Writing	Problem of some magnitude if the affected side is the dominant side	Hilleboe, 1955;
Wernecke's aphasia-lacks internal mechanisms to monitor own speech output	If patient shows personality change, often in the direction of becoming angry with those around him-results from problem	N. Geschwend, 1970;
Aphasia-technical name given for interference with the comprehension and use of language	May be able to understand but not to speak or write by end of 3rd month know patient's speech and language capacity.	Strokes, a guide for the family, 1969; Aphasia and the family, 1965; J. Eisenson, 1973; J. E. Sarno, Jr., 1967 N. Geschwend, 1970; Cerebral Vase Dis & Stroke, 1972; L. Galton, 1975; Understanding stroke 1972; Taylor, 1962;
Speech problem	over 1 million hemiplegics in U.S.. Hemiplegics have speech problem.	
Aphasia with aproxia of speech	Frequently occurs with aphasia. Patient appears to have suddenly and strangely lost control of his lips and tongue. Mouth not paralyzed	J. S. Keenan, 1974; Taylor, 1962;
Aphasia with auditory agnosia	Patient often acts as if he had a hearing loss. Hearing loss does not seem to be total, degree of loss seems to change	J. S. Keenan, 1974;

TABLE 1-8, CONT'D.

Problem	Dynamics	Source
Aphasia with confusion or anxiety	Aphasic patients typically confused for a few days after onset of the brain damage, most anxious occasionally about condition. Some patients' confusion persists.	J. S. Keenan, 1974;

V. Functioning as a Member of Society

The literature on stroke provides information about functioning as a member of the labor force, as a member of community, and as a member of the family.

VI. Functioning as a Member of the Labor Force

Table 1-1 summarizes retrospective study of male members of the labor force in the 45-64 age group (which is considered the most economically vulnerable through the loss of income). Company A in the study represents a large urban service company which is representative of light industry in the U.S. Company B represents heavy industry whose work entails more physical effort, representative of a much smaller portion of U.S. industry.

If one disregards the deaths, the proportion of those who returned to work to those who retired is quite similar for the two companies. The difference in the proportion of deaths and survivors in each company could readily have occurred by chance (Ladd, 1962).

Figure 1-1 shows the life-table treatment of employment attrition resulting from disability and death in those returning to work. This rate for all 45-64 year-old males in Company A (Control Group) is included for comparison with the Company A cases, but, of course, cannot be considered a control group for Company B cases. The other data in this figure are based on 50 cases from Comapny A and 23 from Company B followed for 1-5 years. The difference is statistically significant between Company A cases and control. Although the total of patients is rather small in each company, the difference between companies in the proportion remaining at five years is also statistically significant. This suggest that within the parameter of the study the disease pattern measured is not precisely the same in both companies. It can be seen that the greatest rate of attrition in Company A occurs in the first 2 years after which the loss is less precipitous, suggesting that Company A may be able to offer an earlier trial of work to handicapped employees, a measure which is likely to be precluded in a heavy industry such as Company B, with fewer clerical or other light work jobs available. As a result, the cases lost very early represent 'active' cases, such cases in Company B being eliminated in a more extended period of absence from work which

TABLE 1-9

CEREBROVASCULAR DISEASE IN 45-64 YEAR OLD MEN DURING 1955-1959

Company	Total	Cases per 1000 employees aged 45-64	Returned to Work	Disability Retirement	Death	Death Rate per 100,000[1]	Average days lost from work per case	Average Age at onset
A:	108	1.37	50	33	25	40.3	79.2	57.2
B:	51	1.22	23	11	17	45.5	122.6	57.9
		Probable Cerebral Embolism Cases which were Excluded From the Study						
A:	17		7	4	6			
B:	3		1	0	2			

Source: Ladd, 1962

[1]Includes the embolism cases

Note: all cases having disease likely to produce embolization were excluded since the crebrovascular effects are secondary manifestation of primary disease occurring elsewhere.

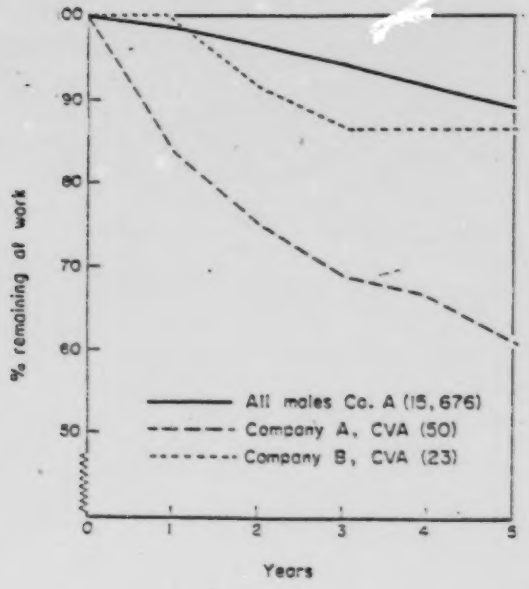

Figure 1-1. Life table treatment of employment attrition resulting from disability and death in those returning to work.

Source: Ladd, 1962

was on the average, 50% longer than in Comapny A. The slightly lower incidence of cases in Company B and the apparently milder course in the survivors able to return to work may reflect the sparing action of work involving increased physical activity suggested by Morris et. al. in their study of CHD in London transport workers.

Table 10 summarizes the complication involved in the CVA patients returning to work. Approximately the same proportion of this category in each company had some neurological defect persisting at the time of return to work and a similar proportion in each had hypertension. Of the 28 in Company A who had hypertension, 9 proceeded to a disability retirement in the follow-up period. This proportion (32%) is approximately the ratio of the disability retirements in the non-hypertensive group (23%), suggesting that hypertension offered little increased hazard. Most of the hypertensives were on anti-hypertensive medication. The disability retirement cases following return to work represent progression of the disease process, inability to cope with the physical handicap, or other diseases. Two of the 3 subsequent deaths in Company A were ascribed to myocardial infarction and one to a second cerebrovascular accident. There was one death in this group in Company B due to a CVA. Ten of the cases in Company A had a second CVA, after which two were able to return to work a second time. There was only the one fatal recurrence in Company B.(Ladd, 1962)

Generally speaking, from a productive employment situation, when a CVA patient returns to work, the employer is often faced with a slow, one-armed person, with less than facile speech, with less emotional control, and perhaps with other physical and mental incapacities in a competitive, speed-conscious, 2-armed, rather unconcerned world (Whitehouse, 1963). The family of a CVA patient should maintain contact with the employer during the early phase of the illness. When return to work may be realistic, the patient should be encouraged to discuss his limitations and a change of hours or position with the employer (Waite, 1975).

TABLE 1-10

SUBSEQUENT FATE AND COMPLICATIONS OF CASES RETURNING TO WORK

	TOTAL	DISABILITY RETIREMENT	DEATH	DIASTOLIC B.P. 90	RESIDUAL NEUROLOGICAL DEFECT	CVA
Company A						
1955	13	6	1 (CVA)	8	6	4
1956	10	3	0	3	5	1
1957	11	4	1 (CHD)	6	2	5
1958	8	0	1 (CHD)	6	5	0
1959	8	1	0	5	4	0
Total	50	14	3	28	22	10
Company B						
1955	5	1	0	1	3	0
1956	4	0	0	3	2	0
1957	4	1	0	1	2	0
1958	7	0	0	7	1	0
1959	3	0	1 (CVA)	0	1	1
Total	23	2	1	12	9	1

Source: Ladd, A. C., 1962, p. 988

Functioning as a Member of the Community

In the past the lack of attention on the part of the community to a patient who had suffered a CVA was caused in part by a combination of fear, ignorance and resignation on the part of all concerned. Because of the overall negative approach to a stroke patient, there were many hidden cases which led to a problem in collecting statistics as well as contributed to the development of a stigma regarding stroke (Wright, 1955; Whitehouse, 1963). In recent years, a significant change in attitude has taken place in the community as a whole and in physicians in particular, toward the care of chronically sick and disabled citizens. This is a result of general acceptance of the concept that our obligation and responsibility to handicapped individuals is not met until they are restored to the fullest possible degree of contented and productive living (Piersol, April & June, 1958). There is a growing recognition that the community must help by reducing or removing architectural barriers and insisting on their exclusion in new construction. The community should offer social and recreational opportunities for those hemiplegics who can travel to them. Even though very few hemiplegics will work again, there should be opportunities for work or at least a sheltered workshop. Voluntary agencies complete the picture of a world suitable for a stroke victim. To those who object to the cost and effort of stroke rehabilitation because of the slow and minor gains and therefore insist that hemiplegics should be left along to die with dignity, it should be pointed out that there is nothing dignified about dying by surrender and neglect but much that is uplifting and rewarding about trying to live like a human being (Light, 1975).

Many people feel uncomfortable and inadequate in relating to a patient with a language problem. Both the patient and friends should be encouraged to

socialize. The family can help make the visit better by discussing the situation with the visitor beforehand so he knows what to expect. If the visit is not handled properly, the visitor may be reluctant to call again and the patient may refuse other socializing, visits or outings (Horwitz, 1962).

Thurner (1973) explores communication problems by dramatizing a case involving a stroke victim deprived of speech but not of rational thought. An attempt is made to view the problem from the positions of both the patient and the therapist. Problems of public attitudes and the effects of isolation and of independence are also discussed in this report.

In his personal account of life after a stroke, McKenzie Buck (1957) comments that one of the main aspects that seemed to help combat depression was that the normalcy of the home situation did not change significantly as a result of the accident. "A great deal of conversation was accomplished, at least on the part of visitors, which assisted me in orienting myself regarding life as it was then progressing. The best treatment, I am sure that I acquired, was frequent visitations with my neighbors and professional colleagues."

Functioning as a Member of the Family

Stroke is a family illness. The sudden and disruptive onset of a stroke constitutes a social and emotional crisis. It is frequently accompanied by increased tension, guilt or hostility, cognitive and perceptual disorganization and confusion for those involved. Stroke can represent a threat to the patient's and the family's needs, a loss or deprivation, and a challenge to problem solving mechanisms. It may also revive unresolved feelings about the past illness of the patient or other family members. When there have been estranged or conflicting relationships between family members, considerable expressions of guilt about the cause of the illness may be expressed. These feelings may be compensated for by the family member being over-protective and indulgent of the patient. Conflicting relationships prior to the stroke may result in the family's avoidance of the patient and staff after the stroke or even in extreme cases may lead to total rejection of the patient or responsibility for participation in his care. For families in which the patient has been a dependent member or scapegoat, this increased disability and dependence may be intolerable and be the stimulus for a separation from the patient. Where there have been close and positive family relationships, there may be a total denial of the disability or an overindulgence of the patient's desires and reinforcement of his dependence.

A frequent response to stroke in a family member is that other members of the family may become sick physically, emotionally or empathically (James, 1970). Those who become physically ill seem to resist diagnosis or treatment until the stroke patient begins to improve or until he is rehabilitated. Some family members may exhibit intense depression, a phobic reaction to hospitals and intensification of emotional instability necessitating resumption of psychiatric treatment or the use of medication. These types of family responses to the stroke may reflect their values concerning health, illness and body image (Waite, 1975).

All the efforts of the entire rehabilitation team will be fruitless if the patient is not made to feel that he is wanted particularly by immediate members of the family. The patient will tend to give up if he comes to realize that his ever-loving wife no longer wants him at home. In some cases it is impossible for the patient to receive proper care at home but in so many cases, the wife actually abandons the husband, or the husband abandons the wife, because the partner is no longer able to provide the companionship to which she or he has been accustomed. This is why it is so important to take partners into confidence and let them participate in the rehabilitation program from the very beginning. Often when the wife is convinced by actual observation that the patient, although handicapped, will not necessarily be an undue burden, she will accept his return to the home more readily (Zankel, 1969).

It is important that the family group seek appropriate professional people to assist in formulating a program of retraining. This move should be made during the first week following the attack. In this manner then, the family will receive training in preparation for the patient's return home. Persons who need to be contacted are those in medicine, psychology and clinical speech. This group may also be of assistance to the individuals' former employer and working environment (Buck, 1957). The family of the patient should transmit their faith verbally because even though the patient may not fully understand the words, he will comprehend the attitude if he is talked to with expressive facial and bodily movement, touched with reassurance and made to feel that he is cared for. The key need is for a receptive, motivated family or at least one that can be encouraged to accept reasonable responsibility (Whitehouse, 1963).

There is little argument that the home is the best place for the stroke patient even if it is only minimally adequate. Sometimes during the early part of

a patient's recovery, well-meaning relatives induce the spouse of the stroke patient to move into their home (where there may be more room for the patient) and to give up a home which may have great meaning. Both by implication and in actuality, this kind of precipitous planning can have a disastrous effect on the patient and his future social recovery (Cockerill, 1963). In preparation for the return home of a stroke patient, the family should correct such hazards as loose scatter rugs, slippery stairways or poorly lighted halls and installing such aids as handrails, grab bars, toilet siderail, bathtub seat, rubber mats, or any number of other devices for the safety and convenience of a disabled patient (Guidelines to stroke-patient care, R.N. Magazine, September, 1975).

The stroke patient's resocialization and re-integration into the family and community is an important adjustment for both the patient and his family. The process provides the afflicted person with opportunities for experiences that enable him to regain confidence and respect for himself despite the presence of physical disabilities or language disorders. It engenders the potential for a stroke victim to visualize himself as a person again rather than a patient (Hackler, 1971). Periods of depresssion are common in the patient and are a natural reaction if they are not too deep or prolonged. Because progress is slow, patients may become indifferent to re-learning skills they once took for granted. The stroke patient is apt to be sensitive about his condition and may grow suspicious of what people are thinking about him. At times like this, he needs constant encouragement to overcome his depression and face up to his problems (American Heart Association, 1969).

The family of a stroke patient may begin his rehabilitation by encouraging his contribution to the home in minor ways. Such acts lend more of a feeling of worth and status, of earning his keep in a small sense and lead to a growing feeling of independence in the patient. The patient should be left alone at times as this will relieve the family and also help the patient to gain confidence and independence (Horwitz, 1962).

The National Easter Seal Society for Crippled Children and Adults has made available a publication entitled Understanding Stroke. Some of their recommendations for the rehabilitation of the stroke victim which may be used by his family are listed here:

Do not correct mistakes in speech or writing of the stroke patient. Say what you think he is trying to say and ask him if you understand correctly.

Do not talk for him if he has trouble speaking. Ask him a question which he can answer by nodding "yes" or "no".

Do not interrupt him when he is trying to speak. Give him your whole attention. Speak in your natural voice. Shouting won't help to make the stroke patient understand.

Never talk about the stroke patient in his presence. He may not answer, but he probably understands.

Do not confuse the stroke patient with too many people around him at one time. Make visits short and cheerful. Encourage him to go out, but do not push him into going.

Do not forget to stand at his "good" side if he has a loss of half of the visual field. Place yourself where he can see you.

Do not be alarmed, embarrassed, or afraid if stroke patient cries, acts depressed, or swears. Be casual about his behavior. Treat him with courtesy and respect.

See that the stroke patient practices and performs his exercises faithfully.

VI. Technologies

1. Surgery--Some types of stroke can be anticipated and prevented by surgery. Carotid (neck) arteries filling with atherosclerotic deposits can be diagnosed by X-ray. A blocked neck artery can be cleared by surgical removal of the block. Implanted synthetic material can be used to bypass the blocked segment, providing adequate blood supply to the brain (American Heart Association, 1974 Heart Facts).

Cerebral angiography patients were screened in one clinic and it was found that 982 out of about 2,500 cases were potentially curable by surgery. Of these 982, approximately 92% were cured or improved. Operative mortality was only 6% (National Heart Institute, 1969). Other articles which discuss surgery are (Freed, 1975, pp. 146-170; Nash, 1966, In Hospital Based Stroke Program, Kansas, n.d.).

2. Electronics and related technologies have joined forces with medicine to introduce a wide variety of devices and techniques that make more precise and effective the diagnosis and treatment of cardiovascular disorders:

Cardiac catherization	Radioisotope scanning
Chemical treatment	Computers
Angiocardiography	Ultrasound
Vectorcardiography	Electrical aids
Phonocardiography	

(American Heart Association, 1974 Heart Facts; Zankel, 1971, p. 138; Liberson, 1975, p. 483-493)

3. Drugs--Anticoagulant drugs must be sharply limited to specific categories of intracranial vascular disease (Millikan, 1963; Modern Medicine, May 5, 1969). Contraindications for anticoagulants: Heparin, antidote intravenous protamine sulfate, long termanticoagulation - Warfarin, Vit K'.

4. Diagnostic Aids--

Lumbar puncture

Ophthalmodynamometer - retinal artery press

Endarterectomy

Arteriography

Doppler instrument

Use of CO_2, papaverine, amyl nitrate (Fryer, et al., The Mason Clinic, Seattle, n.d).

Vasodilating agents

Thrombolytic agents

Carotid compression tonography (CCT) (Galton, 1975).

Intracarotid Amytal Studies (Wada test)

Broadbent (1954) technique (Geschwind, 1970)

General Neurological Tests

Neurovascular tests

Differential Diagnosis

Angiography (Toole, 1968; Benton, 1970; Fields, 1974; Resch, 1969)

5. Therapy--pharmacological, surgical, rehabilitative (Mancall, 1975)

Treatment of hypertension

Physical therapy - passive motion of the limbs and active exercises (American Heart Association, 1969)

Walking preparation and gait training (Brunnstrom, 1970)

Sinusoidal stimulation - it has been shown that this may be used as an assistive device to improve motor function in selected cases of stroke paralysis (Zankel, 1960; Hilleboe, 1955; Brown, 1963; Kitowski, 1970; Kent, 1971)

Range of motion exercises - relieve muscle ache, promote circulation and relieve tension (McNeil, 1975)

6. Rehabilitation-- Leg brace, cane, platform orthosis, dynamic orthoses, footboard, trochanter roll, catheters (Kitowski, 1970).

Diet, Vocational Rehabilitation, Hypnosis, Specific techniques for re-learning to eat, dress, make bed, use bathroom, garden, work in household, workshop and miscellaneous tasks (Lombardi, n.d)

7. Self-help technologies--Wheelchair, pick-up walker, braces, 4-legged cane (Gordon, 1973)

Trapeze - to help patient sit up in bed

Clothing - without too many fastenings

Knife - that anchors meat and cuts by rocking to assist in eating

Washing - hand brushes which fasten to the top of wash stand by suction cups

Shower or tub - with steps, seat, handrail

Walking canes - with suction cups

Shoes - snow cleats in winter

Kitchen gadgets

Telephone cradle

Typewriter - with one hand keyboard (Hilleboe, 1955)

Hand rails

Rubber mat, seat in bath

Soap in wash cloth pouch

Board table for wheelchair

One arm card holders

Bed side table - bell within reach (American Heart Association, 1969).

VIII. Service Delivery

In a report by the Rehabilitation Study Group on Stroke Rehabilitation, it is suggested that community health services should be available to those who can not travel. This service should include a visiting physician service (family physician/internist and other specialists) for both regular follow up and emergency care. The nurse, physical therapist, social worker or family should have the freedom to call in the physician between visits if the patient's condition appears to warrant medical attention. Community health services should also include:

1. Mobile X-ray and laboratory units when such services are ordered by doctors.

2. Dietary counseling when needed.

3. Homemaker or household assistance when indicated.

4. Delivery of nutritionally balanced meals for the lone, isolated stroke patient unable to cope with such matters as shopping and cooking.

Stroke Center - part of progressive care center of hospital (Davis, 1970).

Stroke Clubs - how to organize (National Easter Seal Society for Crippled Children and Adults, 1972).

Visiting Nurse Association	Rehabilitation Center
Department of Vocational Rehabilitation	Social Services
Dept. of Social Welfare	Medical consultation
Aid to Disabled	Training for activities of daily living
Old Age Security	Vocational Training
Family Social Services	Driver's Training
Local Heart Association	Psychological services
County or State Health Department	Pre-employment training and sheltered workshop placement
Red Cross - assit with transportation	Day care program - Rehab. Center

(Hackler, 1971; Easter Seal Society, 1963; Kitowski, 1970).

Outside of Rehabilitation Centers, the Easter Seal Societies have services and itinerant therapy programs (bringing treatment into the home); equipment pools which loan out for temporary use such items as braces and other appliances; speech centers; workshops (offer renumerative employment opportunities adapted to the individual needs of clients) job placement; sales outlets, which help market products made by the home-bound; camping programs, which provide social and diversional activities adapted to individual abilities; referral services, which guide the patient and his family in utilizing other resources available in the community; "friendly visitors" services, which bring volunteers into the patient's home to alleviate the tedium of convalescence; continuing efforts to eliminate architectural barriers in buildings used by the public.

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CHAPTER 2
DISABILITY ANALYSIS: SPINAL CORD INJURY

I. Definition

Ludwig Guttman (1971) characterizes spinal cord injury as being "one of the most devastating calamities in human life". The spinal cord is the primary transmitter of all messages to and from the brain, controls voluntary movements, proper bladder, bowel and sexual functioning, and the autonomic nervous system which is responsible for respiration, heart regulation, and blood circulation as well as other body systems. Therefore, a "severance or severe injury of the spinal cord... always results in a disablement of great magnitude." (Guttman, 1971)

Individuals with spinal cord injuries are generally classified into two broad categories: paraplegics and quadriplegics. An individual termed paraplegic, exhibits paralysis of both legs and the lower part of the body due to injury (from a variety of etiological factors) of the chest (thoracic) and the lower back (lumbar) segments of the spine. An individual termed quadriplegic, exhibits paralysis of the legs and arms plus the lower part of the body due to injury (from a variety of etiological factors) of the neck (cervical) segment of the spine. Both forms of spinal cord injury result in the partial to complete loss of voluntary movement and sensation; in both there is some trunk involvement, unless the injury is below the lumbar 1 vertebra; and in both types there is some impairment of the body functions which are dependent upon the autonomic nerve fibers (Felton, et al., n.d.).

The more "superior" (toward the head) the injury, the more extensive the damage to the body systems. Therefore, if one injures

the cervical portion of the spinal cord, there will be more extensive damage to the body (i.e. an individual will become quadriplegic) than if one were to injure the thoracic portion of the spinal cord (one would become paraplegic). Furthermore, it is the level of the spinal cord rather than the vertebra (bone) injury which is important. According to Freed (1965) traumatic quadriplegia is the "...end result of injury to the cervical portion of the spinal cord in the form of concussion, contusion, compression, laceration, puncture, or transection, with or without fracture or dislocation of the osseous (bone) structures of the cervical spine." Thus, two patients with a C6 vertebrae injury will have different capabilities if the cord damage in one is above the C6 vertebrae and damage to the other individual is below the C6 vertebrae (Singh and Magner, 1975).

The functional limitations of the spinal cord injury are dependent not only upon the location of the lesion but also whether or not the lesion is complete or incomplete. For example, complete severence of the spinal cord results in paralysis with loss of sensory and motor functioning below the level of injury (Kraus, et al, 1974). The completeness of the lesion also plays a crucial role in the life expectancy of a spinal cord injured individual (O'Connor and Leitner, 1971).

The quadriplegic is potentially more functionally disabled than a paraplegic. In fact, the quadriplegic condition is used as a general reference for the term "severely physically disabled". The criteria for a severely disabled individual include:

1. confined to a wheelchair.
2. unable to transfer between chair and bed, etc. without some assistance, human and/or mechanical.

3. weakened trunk and arm movement.
4. partial or complete loss of hand movement.
5. partial or complete loss of bowel and bladder control.
6. cannot live alone or care for his/her many physical needs independently.
7. requires some degree of skilled medical supervision to sustain health. (Maxwell, 1971).

Fifteen years ago, the medical profession usually was unable to save lives of patients with high (cervical) spinal cord injuries (i.e., quadriplegics). However, with the recent advances in techniques and new procedures within medicine, the potential quadriplegic is being saved (Maxwell, 1971). According to Rusk (1964) "...with the present rehabilitation techniques and patients perseverence and motivation, the majority of these patients can live dignified and productive lives." Furthermore, although the quadriplegic is potentially more severely disabled than the paraplegic, many factors (i.e. personality, family support) intervene which influence the rehabilitation outcome. And thus, one cannot predict with complete certainty that a quadriplegic will be more severely disabled than a paraplegic.

II. Societal Characteristics of Persons with Spinal Cord Injury

The literature documents the prevalence, incidence, mortality, and costs of spinal cord injury.

Prevalence

Accurate prevalence figures are unavailable at this point in time. Singh and Magner (1975) cite a rather low prevalence figure of 60,000

to 100,000 individuals. However, other sources tend to support a statistic of approximately 150,000 individuals as a fairly reliable figure. Figures compiled by the University of Colorado (1962) and Meyer (1973) indicate that there are probably more than 150,000 quadriplegic and paraplegic individuals in the U.S. Furthermore, according to Meyer (1973) the numbers are steadily rising.

In 1971 the Dept. of H.E.W. (1973) gathered data which indicate that 145,000 to 170,000 persons have complete or partial paralysis due to traumatic spinal cord injury, a rate of 0.8 per 1,000 individuals. These statistics include individuals sustaining acute traumatic lesions, but do not account for individuals suffering from spinal cord injury due to degenerative or chronic conditions. HEW compiled its figures from statistical summaries limited to persons admitted to spinal cord centers or hospitals. These summaries were gathered from the following sources: Wilcox, et al., 1970; Key and Retief, 1970; Meinecke, 1968; gehirg and Michaelis, 1968; Chesire, 1968. Therefore, these figures can be considered to be on the low side, since individuals sustaining SCI due to etiologic factors other than trauma and/or dying before admittance to care centers are not incorporated within the prevalence figures.

Incidence:

There is some disparity in the incidence figures for spinal cord injured individuals in the United States. The method of data collection is the major reason for variety in these statistics. Most studies utilize only hospital records and do not take into account those sustaining

spinal cord injuries, but dying at the scene of an accident. The following three studies are cited as examples. It should be noted these figures were complied by specific geographical areas within the U.S., rather than being overall figures for the entire U.S.

Author	Incidence Rate	Geographical Area
Sharman and Owen (1970)	12.4 per million	New England
Wilcox, et al (1972)	25 per million	Hawaii (1965-1970)
Young (1974)	35 per million	Arizona (1973)

According to Meyer (1973), the data indicate that the incidence of spinal cord injury varies by region and/or state. Hawaii's rate varies from 35 to 50 per million; Nevada reports 25 per million; and the Chicago area has a rate somewhere in the 50's, possibly due to the crowded highways and the area traffic patterns.

A number of other researchers have compiled data on overall incidence figures in the U.S. However, the methodology utilized was not specifically stated. According to Young (1970), there are approximately 4,000 to 10,000 cases per year of spinal cord injury. The University of Colorado (1963) notes that traumatic lesions of the spinal cord strike between 4,000 and 5,000 persons per year; while Singh and Magner (1975) report a figure of 4,500 to 10,000 new cases per year. The Dept of HEW (1974) estimates between 10,000 and 12,000 new cases annually of spinal cord injury.

Recently, Kraus and his associates (1974) conducted a study of an 18 county area in northern California. One of the tasks of the study was the compilation of accurate incidence figures. Statistics were compiled for a two year period, 1970 and 1971. It was found that

619 persons among 5.8 million (29% of the state's total population) suffered some form of spinal cord injury during 1970 and 1971. This results in an incidence rate of 53.4 per million. The percentage occurrence of incidence cases was about the same for each of the two years under study (1970 = 49.3%; 1971 = 50.7%).

The materials used to collect the date included not only hospital admittance records, but coroner's office records and the records of the State of California Departments of Health (Crippled Children's Service); Rehabilitation; and Industrial Relations (Workmen's Compensation). The researchers recorded only those injuries which were the result of acute traumatic lesions. The average annual incidence rate, 53.4 per million population, was considerably higher than rates previously collected in a number of other studies (see table above). If the criterion of incidence as defined by Young (1974), and Sharman and Owen (1970) and Wilcox, et al. (1972)--i.e., persons admitted to a hospital with paralysis due to spinal cord injury--is applied to the data collected in the Kraus, et al. (1974) study, an incidence rate of approximately 32 per million results. This rate is similar to that reported by Young (1974) for Arizona and by Wilcox et al. (1972) for Hawaii, but is still three times higher than that found in New England (Sharman and Owen, 1970). According to Kraus et al. (1974), an estimated 11,220 individuals will annually incur traumatic spinal cord injuries in the United States. About 4,200 of these will die before they reach the hospital for treatment; and an additional number (1,150) will succumb during the period of hospitalization.

Mortality:

A. General Trends:

It has been estimated that over half of the patients with spinal cord injuries never survive long enough to be admitted to the hospital. (Meyer, 1963). Meyer (1963) believes this is partially the result of improper patient management: e.g. delayed attention, ineffective handling, lack of awareness of the needs of the spinal cord injured, slow transportation, inept personnel at the initial stage of hospitalization and delayed referral to the proper specialists.

According to Meyer (1972), approximately 2,000 to 3,000 people will die each year of spinal cord injuries. It has been estimated that 4,200 individuals of a total of 11,200 new cases per year will die before they reach a hospital for treatment. An additional 1,150 will succumb during the period of hospitalization (Kraus, et al.,1974). Kraus et al. (1974) reports that among 619 cases of spinal cord injury in an 18 county area of northern California, there were 299 deaths, indicating a 25.8 per million population mortality rate and a case fatality rate of 48.3%. Included within this figure were deaths occurring during the period from injury event to hospital discharge. Approximately 79% of the 299 deaths were dead at the scene or dead-on-arrival, while the remaining 21.4% died while hospitalized. An analysis of the causes of death reveals the following:

187	(62.5%)	spinal cord injury was the primary cause of death.
37	(11%)	spinal cord injury was a significant condition in the cause of death.
75	(26%)	death the result of massive multiple injuries, including spinal cord injury (i.e. whole body crushed).

It has been found that the survival rate of a spinal cord injured individual is related to the level of injury and the completeness of the lesion (O'Connor & Leitner, 1971). If the survival rate is compared to the level of injury, individuals suffering from cervical injuries have shorter life expectancies (Ruge, 1969). Furthermore, Kraus et al. (1974) found survivorship for quadriplegics was poor regardless of etiological factor. And according to O'Connor and Leitner (1971), 30% of all quadriplegics succumb during the first 30 days. A comparison of the survival rate of quadriplegics vs. paraplegics indicates the latter have a much higher survival rate than the former:

Patients	Quads*	Paras*
1. Discharged Inpatients 1970-71 (116 cases)	27.2	72.8
2. Cases in Southwest Region System (303 cases)	34.4	60.4
3. Berrie, Liberty Mutual Insurance Co.	30.0	70.0

* by percent of total spinal cord injury cases

Source: adapted from Young (1972).

B. Life Expectancies:

According to Dreed (1967), the average life expectancy for cervical spine trauma is approximately 7 years. Seventy-seven percent of those individuals in the 15-24 year age group, surviving the acute phase of cervical trauma, live an additional 10 years; fifty-five percent of those individuals in the 25-34 year age group, surviving the acute phase of cervical trauma, survive an additional 10 years (O'Conner and Leitner, 1971). On the other hand, a paraplegic can expect to live an

additional 20 to 40 years after the onset of the injury (Young, 1970).

Pearce, Perkins and Holladay (1964) reviewed the survival rates of 35 patients. It was found that 32 patients survived their injury for longer than one year (10 quadriplegics and 22 paraplegics). The average survival rate for all patients (N=35) was 11 years and 9 months, and for those living over one year, 12 years and 10 months. The average annual mortality rate was 3.5% higher than the normal population.

C. Comparison of survival rate by etiological factor.

The percentages of those with spinal cord injury surviving the acute period of hospitalization was generally uniform for all external causes, with the exception of recreation-related injuries:

motor vehicle	= 81%
falls	= 83%
firearms	= 85%
recreation	= 95%

However individuals sustaining cervical lesions which resulted in quadriplegia, had a poor survival rate regardless of the external cause of the injury (i.e. motor vehicle--29%, falls--30%). Individuals sustaining cervical lesions which resulted in quadriparesis (partial paralysis of all limbs) had a much higher survival rate. Those sustaining quadriparesis due to motor vehicle crashes and those injured from falls, had a survival rate of 78% and 75% respectively; while ninety-six percent of those with a diagnosis of quadriparesis from a fire-arm inflicted injury survived (Kraus et al., 1974).

D. Case-fatality rate.

In the Kraus et al. (1974) study, percentage case-fatality rates (individual died immediately after sustaining injury) for motor vehicle, fall, firearm, recreational accidents, and all remaining causes were 58.7, 30.3, 54.1, 4.7 and 48.6 respectively. Therefore, it was found that recreation-related spinal cord injuries were more often nonfatal (only 4.7% case-fatality rate) than injuries caused by other factors.

E. Causes of death other than spinal cord injury.

If an individual survives the initial stages of spinal cord injury, the following complications contribute to his/her death: the primary cause of death among quadriplegics is renal failure secondary to urinary infection (O'Connor and Leitner, 1971; Ruge, 1969). Furthermore, respiratory death secondary to pneumonitis and/or pulmonary embolism is not uncommon (Travis, 1961; Bors, 1959; Haas et al., 1965; Montero, et al., 1968).

Among quadriplegics, suicide contributes to a high number of deaths. It may take the form of an overt action, or a condition known as 'physiological suicide', whereby the patient neglects his personal and medical care and thus is at the mercy of decubiti, recurring infections and respiratory and cardiac failure (Kantrowitz and Hald, 1966).

Costs:

It has been estimated that the lifelong cost for the care of a spinal cord injured individual will range from $250,000 to $500,000, depending upon the extent of the injury, with the average cost

approximating $400,000 (Young, 1970).

Costs for initial care range from $15,000 to $40,000, with the average figure being $25,000. The $15,000 figure would be for a paraplegic without associated injuries while the cost of initial care for a quadriplegic with complicated associated injuries would be at approximately $35,000 (Young, 1970). If one were to figure an initial hospital stay to be 150 days (a somewhat generous amount), costs would be incurred as follows:

emergency evacuation	$ 500
hospitalization (at $100 a day)	15,000
medical and surgical fees	3,000
equipment	2,000
home modification	2,000
follow-up care the first year	1,000
	$23,500

After initial care, there are additional costs of maintenance (i.e., costs for attendants, custodial care costs), treatment of medical complication, lost income, lost taxable dollars, amounting according to Young to $400,000. If this cost per case is multiplied by the number of new cases reported annually, the costs become quite large. This figure has been estimated to be approximately $2 billion ($400,000: lifelong cost/spinal cord injured individual X 5,000: conservative estimate of new cases/year). The above cost figure has not been adjusted for a forecasted increase in spinal cord injuries, nor does it take into consideration the approximately 100,000 living persons previously disabled by spinal cord injury (Young, 1970)

The following table notes the differences in costs for a paraplegic and a quadriplegic. Except for loss of income, which has been estimated to be similar regardless of the extent of the injury, the

costs for lifetime treatment and maintenance are much higher for quadreplegics than for paraplegics.

TABLE 2-1

COSTS OF SPINAL CORD INJURY: QUADRIPLEGIC VS. PARAPLEGIC. (adapted from Young, 1972)

I. Quadriplegics (with complete, or essentially complete paralysis):	
Initial Hospitalization:	
128 days x $140/day	$ 17,920
Routine Maintenance:	
$1,000/year x 20 years	$ 20,000
Hospitalization for Medical Complications:	
25 days/year x $120/day = $3,000 x 20 years	$ 60,000
Environmental Modifications	5,000
Total Medical Cost (per case)	$102,920
Custodial Costs:	
$6,000/year x 20 years	$120,000
Loss of Income:	
$6,000/year x 20 years	$120,000
Cost of Vocational Rehabilitation	$ 3,000
Total Non-Medical Cost (per case)	$243,000
Total Cost (per case)	$345,920
II. Paraplegics (with complete, or essentially complete paralysis):	
Initial Hospitalization:	
106 days x $140/day	$ 14,840
Routine Maintenance:	
$300/year x 20 years	$ 6,000
Hospitalization for Medical Complications	
15 days/year x $120/day = $1,800 x 20 years	$ 36,000
Environmental Modifications	$ 1,000
Total Medical Cost (per case)	$ 57,840
Custodial Costs	$ 0
Loss of Income:	
$6,000/year x 20 years	$120,000
Cost of Vocational Rehabilitation	$ 1,000
Total Non-Medical Cost (per case)	$121,000
Total Cost (per case)	$178,840

According to Young (1970), a large portion of these costs are due to mismanagement of the spinal cord injured patients. Substantial savings in costs can be made with the proper initial care and follow-up treatment. Many of the complications are preventable and there is an overuse of custodial care. "The waste is the result of inappropriate fragmented unsystemized and unsophisticated administrative and medical management. A combined systems approach would conservatively reduce this cost by 20 to 30 percent."

Currently, the burden of the cost and care for the spinal cord injured individual rests upon themselves and/or their families. For example, 80% of those individuals who are complete or essentially complete quadriplegics will be discharged and return to their homes (Young, 1972). Thus the cost of care for four out of five will be borne by the family and not the taxpayer (Young, 1972). This cost, per-case/per year at home, has been estimated at $2,000 by the National Institute of Neurological Diseases and Stroke (1973). Furthermore, these costs do not include evnironmental modification of the home, which would be necessary to accommodate a wheelchair. Although modifications are initially costly (i.e. approximately $5,000 for 65% of the surviving quadriplegics and $1,000 for 70% of the surviving paraplegics), they will reduce the expenditures for future medical complications and the need for attendant care (Young, 1972).

According to Lowery (unpublished paper cited in the VA report, 1972), 50% of the parents or spouses of spinal cord injured individuals sampled in his study had to seek part- or fulltime employment to meet the added costs of a handicapped member of the family.

As a result of inflation and increased medical costs, it has become impossible financially for a spinal cord injured individual to live by himself (with an attendant) or with his family. Furthermore, maximum welfare payments are too small to cover the costs of nursing home care (Maxwell, 1971). For example, in the state of California, it is estimated that welfare payments amount to $5,000 per year for a spinal cord injured individual (Savino, 1971). Yet, the cost of institutionalization for persons with spinal cord injury who cannot find or have not found appropriate alternatives in the community are exceedingly high. Furthermore, the custodial care costs, especially for quadriplegics, may exceed traditional medical costs (Young, 1972). According to the National Institute of Neurological Diseases and Stroke, the expenditures for a totally disabled individual at a public and/or private institution amounts to approximately $2,500 to $7,500 per year. According to Fay (1975) these costs can run even higher:

> A motel-like cooperative group residence like the Christian League for the Handicapped in Wisconsin costs about $5 a day. A commune-like transitional halfway house for high level quadriplegics like the ones at the Texas Institute for Rehabilitation and Research (TIRR) and the University of Illinois cost about $8 to $15 a day. Nursing homes for those unable to find community-based alternatives

range from $10 to $75 a day. Some persons with SCI occasionally wind up spending years in even more expensive (e.g. $100 or more per day) custodial institutions and "rehabilitation centers" (p. 147).

It is estimated that 35% of all quadriplegics will, at some point in their lives, experience other disabilities which require custodial care or custodial placement (in institutions). The remaining 65% will require some type of attendant care (either in the home or at an institution). The cost for an attendant working an eight hour day, seven days a week (usually required for those quadriplegics having complete or nearly complete paralysis) amounts to $500.00 monthly. This would amount to a cost, over a 20 year survival period with mean onset at 31, of approximately $120,000 (Young, 1972). These data suggest that among 20 disabilities, spinal cord injury ranks as the highest in terms of the average per year per case costs (Fay, 1975).

In considering the monies for rehabilitation of the spinal cord injured, one is again staggered by the high costs. According to Savino (1971):

> "The projected average cost per rehabilitation over the life of the case is over $12,000. This is much higher than the average cost per rehabilitated client in California. Without rehabilitation, however, the welfare costs for such a catastrophic disability are also very high and average over $5,000 per year. Thus, although it is a relatively expensive investment to rehabilitate these clients with long-term plans, because of the very high welfare costs, there are great fiscal benefits to be derived."

Furthermore, the Liberty Mutual Insurance Company has found that 30% of the spinal cord injured (both quadriplegics and paraplegics) individuals handled by the company have returned to employment, even though insurance benefits resulted in sufficient financial support without working (VA report, 1972). Yet upon returning to work, the average loss of income for each spinal cord injured person is projected to be $6,000 annually (Young, 1970).

According to one state vocational rehabilitation counselor, "Quads are expensive, they use up case service dollars that I do not have enough of anyway" (Fay, 1975). Poor integration of services has resulted, in many cases, in increased overall costs in the rehabilitation of the spinal cord injured. For example, the failure to approve state or federal dollars for environmental modifications in the work environment may result in later costs for lifetime custodial care due to inaccessible home and/or work environments (Fay, 1975).

III. Demographics

A. Sex

Statistics collected from a number of sources indicates that the majority of individuals incurring spinal cord injuries are male. According to Young (1970), approximately 70% of those injured yearly are males. Singh and Magner (1975) cite a statistic indicating that 78% of those individuals suffering spinal cord injuries are male. Kraus et al. (1974) conclude that incidence rates for spinal cord injury are three times higher for males than for females.

B. Age

Males aged 15-24 accounted for the single largest category of persons with spinal cord injury. The peak incidence rates were found to be 20 to 24 years of age for males and 25 to 29 years of age for females (Kraus et al., 1974). The incidence of spinal cord injury increased sharply after 15 years of age among males, declined after the age of 30, but tended to increase gradually through the latter decades of life. "The incidence of spinal cord injury peaked about five years later for females, and there was no peaking in rate at age 35-44, as observed in males" (Kraus et al., 1974).

Results from the Kraus study indicate the following: Motor vehicle collisons resulted in the highest injury rates for all age groups. However, in the 15-24 age group, high injury rates were not only due to motor vehicle collisons but were the result of firearm and recreational related accidents. The highest incidence of injury among persons 55 years and older was due to falls.

C. Ethnicity

The Kraus study (1974) indicates that the incidence of spinal cord injury in highest for blacks and lowest for Japanese, Chinese, American Indian, and Filipino groups. The relative rate of incidence of injury (the ratio of the rate for each race/ethnic group to that of the white group rate) for blacks (both sexes) was twice that for whites (both sexes). The injury rate for persons in the "other" category (Japanese, Chinese, American Indian and Filipino) and the Mexican American race/ethnic category was approximately one-half the

rate for persons in the white group. "This high injury rate for blacks (165.8 per million for males and 44.1 per million for females) could not be attributed to a disproportionally large representation of young persons in the black population" (pg. 16).

A comparison of incidence rates by sex among the various ethnic groupings indicates that for some groups there are significant differences. The risk of injury is 13 times greater for male Mexican-American males than for Mexican-American females. The risk of injury was 3.7 times greater for black males than black females, while among whites, the risk of injury was 2.5 times greater among males than females. No significant difference between males and females was found in the "other" ethnic category (Japanese, Chinese, Filipino, American Indian). Furthermore, it has been found that the causative agent differs by ethnic group. Among black, an excessive number of spinal cord injuries are the result of firearm accidents/crimes, while among the "other" ethnic-race category (Japanese, Chinese, American Indian and Filipino) falls cause a very significant number of injuries (Kraus et al., 1974).

D. Marital Status

Kraus study (1974) reveals the following statistics: The average annual incidence rates of spinal cord injured individuals by marital status were highest among divorced, separated and never married individuals and lowest for widowed and currently married persons. If these figures are differentiated by sex, the findings indicated the incidence rate of injury for widowed males was almost 4 times that for widowed females; while the incidence rate was more than five times

higher for separated males than for separated females.

A study conducted by Kaplan (1966) presents data on the marital status of spinal cord individuals prior to and after the onset of the disability. Sixty-five percent of the males were married prior to the onset of the spinal cord injury, while 48% of the women were married prior to the onset of the disability. After the onset of the injury the data reflected the following: (1) no males previously unmarried were married, but 14% of the previously unmarried females were married. (2) The percentage of males never married was 31% and the percentage of females never married was 38%. Of those males and females married prior to the onset of the disability, 33% of the males and 77% of the females were still married afterward. Sixty-one percent of the males were either divorced or separated and 23% of the females were either divorced or separated.

E. Geographical region

Kraus et al. (1974) could not find a significant relationship between geographic area and the occurrence of spinal cord injuries. However, according to Meyer (1973), the incidence of spinal cord injury varies by region and/or state. Hawaii has a rate of between 35 and 50 per million, Nevada reports 25 per million, and the Chicago area has a rate somewhere in the 50's, possibly due to the crowded highways and the area traffic patterns (Meyer, 1973). Sharman and Owen (1970) report a 12.4 per million incidence rate for the New England area.

Findings indicate that most frequently pedestrian injuries due to automobile accidents and injuries caused by falls occur among individuals living in crowded environments (urban areas). The more crowded the conditions, the more children playing in the streets, and it has been found that teenagers with no other place to play other than the streets have a disproportionally higher rate of spinal cord injuries in the urban areas than the rest of the population. This appears to be a worldwide phenomena (Hall et al., 1972; Howard et al., 1971).

IV. Etiology

The most common cause of spinal cord injury is trauma (Felton, Perkins and Lewin, n.d.). Sixty to eighty-five percent of persons hospitalized with spinal cord injuries have experienced an acute traumatic episode (Kraus et al., 1974). Trauma to the spinal cord is usually due to an accident (i.e., automobile, diving into shallow pools, sports, industrial accidents), though trauma can also result from combat injuries. Additional causative agents include: (1) congenital factors (i.e., severe forms of spina bifida) which sometimes cause paraplegia but rarely causes quadriplegia; and (2) diseases which adversely effect the spinal cord such as cancer and other tumors of the spinal cord, infections and abscesses of the spinal cord, multiple sclerosis, and tuberculosis of the spine (this disease may create transitory or permanent paraplegia).

According to Singh and Magner (1975), automobile accidents are the leading cause of spinal cord injury in the United States. Kraus

et al. (1974) note: "...motor vehicle crashes are responsible for the major forms of impairment coincident with traumatic lesion to the spinal cord." However, increasingly, motorcycle and motor scooter accidents are contributing to spinal cord injuries. Among older persons, falls or just not being cautious enough as pedestrians, result in the majority of spinal cord injuries (Singh and Magner, 1975).

The extent to which the various etiological factors contribute to traumatic spinal cord injury has been analyzed by Kraus et al. (1974), and are summarized as follows:

1. 56% of the spinal cord injuries are due to motor vehicle collisions.
   one-third = single vehicle
   one-third = multiple vehicle
   10% = collision involving a motorcycle
   20% = pedestrian struck by motor vehicles.

2. Falls account for about 20% of the total number of spinal cord injuries.
   75% = due to falls from an elevated place (i.e. stairs, roofs).

3. Firearms = 12% of the cases
   50% = due to homicide or attempted homicide.

4. recreational or sporting activities = 7%
   75%=resulted from diving accidents.

5. Other causes = 6%
   nonfirearm self- or criminally-inflicted injuries
   being struck or caught by an object
   airplane crashes
   military injuries.

Combat injuries, though causing a relatively small number of spinal cord injuries, have been instrumental in causing significant improvements in the care and treatment of spinal cord injury (Singh and Magner, 1975). Meyers has compiled the following date pertaining to the causes of spinal cord injury in three states in the United States. The data

substantiate the contention that automobile accidents cause the majority of spinal cord injuries. It should be noted that statistics on combat-related injuries are not included in these figures.

SPINAL CORD INJURY: 1972

Causes of Injury

	Nevada	Hawaii	California
Auto	55 (53%)	49 (21%)	329 (43%)
Falls	7 (7%)	65 (29%)	92 (12%)
Water Accidents	9 (9%)	31 (13%)	91 (12%)
Gunshot Wounds	10 (10%)	12 (5%)	146 (19%)
Motorcycle	2 (2%)	6 (3%)	45 (6%)
Struck by Falling Objects	3 (3%)	17 (7%)	9 (1%)
Bends	0	8 (3%)	0

According to Meyers (1973), one-third of all spinal cord injuries result from highway accidents; one-fourth occur at work and are the result of a fall or a blow to the back; and one-fifth occur in the home, often resulting from a fall. There have been increases in injuries due to sports activities, and gunshot wounds have also become a more frequent causative agent. In Michigan and Wisconsin, snowmobiles are contributing to increases in the number of spinal cord injured (Meyers, 1973).

The type of functional impairment is significantly related to the external cause of the injury. It has been found that quadriplegia results predominately from motor vehicle crashes, falls, and/or recreation-related injuries, while paraplegia results from motor vehicle crashes or gunshot wounds. According to Young (1971):

> Motor vehicle crashes contribute equally to all types of impairments from spinal cord injury, suggesting that the forces

transmitted in such crashes are distributed broadly over the entire torso and not only to one segment of the spine. This pattern was not apparent for other external causes of injury. For example, among those admitted to a hospital with gunshot injury to the spinal cord, the lesion most frequently resulted in paraplegia while recreation-related injuries (mostly diving) result in a complete or incomplete lesion in the lower part of the cervical spine. Survival rates (those discharged from the hospital alive compared to those admitted with a spinal cord injury) varied according to type of impairment and cause of injury. Poorest survival rates were seen for quadriplegics regardless of external cause of the injury (pp. 25-26).

V. Life Functions

Although the spinal cord injured individual is affected by health problems during the initial period of recovery, the major problems faced by a spinal cord injured individual are emotional, interpersonal, vocational and functional. (Functional problems refer to difficulties with excretory functioning, sexual responses, severe mobility and muscular restrictions.) (Siller, 1969). The common problems facing a spinal cord injured patients include: worries about sexual functioning; the possibility of walking again; future vocational possibilities; fears about going home; fears of discharge; alternatives to permanent hospitalization; financial considerations; family problems; medical problems and the initial reactions of "walkers" (Mann, Godfrey and Dowd, 1973). These are essentially difficulties in the area of interpersonal

relationships and will be discussed in later sections (see Social Attitudinal Life Function and Functioning as a member in the Labor Force sections).

Health:

A review of the literature concerned with health problems of the spinal cord injured reveals a variety of health impairments. The extent to which these complications affect the health of the spinal cord injured varies: some can be alleviated by following a carefully prescribed health regimen; others occur during the initial stages of the injury only; while some problems can be controlled but not entirely alleviated. As noted above, health difficulcies do not constitute the most serious problem faced by the spinal cord injured individual. However, health problems can, and do, cause a certain amount of discomfort, and require prompt attention and treatment so as not to reach a critical stage.

A. Decubitus Ulcers:

Decubitus ulcers (decubiti) form as "the result of unrelieved pressure on the bony prominences of the body while the patient is bedridden, or later while sitting for prolonged periods in a wheelchair" (Felton, Perkins and Lewin, n.d.). Ulcers, of this type, most frequently develop on the ischial tuberosities, sacrum, skull, heels and for persons with a cervical vertebral fracture (who must be kept in a prone position) on the forehead and knees. The occurrence of decubiti can be lessened during the initial stages of injury, by turning the patients in bed every two hours and massaging the skin

in the pressure areas. Wheelchair-bound individuals can be trained to shift their support from time to time to prevent ulceration (Young, 1971; Long, 1971).

B. Autonomic Hyperreflexia:

This state manifests itself in varying degrees of severity. It can range from mild sweating to severe hypertension with an extremely painful headache, massive diaphoresis and a "subjective feeling of impending doom." In its most malignant form, severe hypertension can produce intracerebral hemorrhage and death. The condition is apparently related to the excessive release of norepinephrine at ganglia of the sympathetic nervous system. This release may be triggered by a variety of stimuli, including: skin pressure or stroking, change of position, or the presence of systemic infection; however, the most common cause is stimulation of the bladder through overstretching or during the process of irrigation. The most common cause of severe headache in quadriplegics is probably distension due to a plugged catheter, with resultant autonomic hyperreflexia and hypertension. Relief of the catheter obstruction usually produces an immediate drop in blood pressure and relief of symptoms. However, preventitive measures can be taken to minimize the severity of autonomic hyperreflexia, i.e. irrigations of the bladder should be performed in a sitting position; drugs can be used to reduce bladder sensitivity to distension.

C. Calculi:

Renal, urethral and bladder calculi frequently interfere with the rehabilitation program of a spinal cord injured patient and may also lead to impaired renal function and death. The incidence of renal

calculosis reported by various authors ranges from 1.2 percent to 25.8 percent, with an average of 10 to 11 percent (Bors and Comarr, 1971). The wide range of incidences reported are most likely dependent on the methods used for detection, the enthusiasm of the medical program of urinary care, the adequacy of follow-up evaluations, and the geographic areas which may be endemic for calculi (Stover and Miller, 1975).

The exact cause of "stone" formation has not been identified, however, a number of factors appear to affect the production of calculi: alterations of mineral metabolisim, a neurogenic bladder, recumbency, infection and indwelling catheters. "Infection is one of the most common denominators agreed upon as an initiating factor for calculus formations" (Stover and Miller, 1975). Recumbency (lying in bed) is to be avoided as much as possible, catheters are to be removed as soon as they safely can be and infection should be prevented and/or treated immediately (Long, 1971).

D. Heterotopic Bone Formation:

Heterotopic bone formation results in the laying down of new bone in soft tissues, often around joints or along the femoral shaft. "Although the joint itself is not attacked, limitation of motion often occurs as a result of encroachment by the extra-articular formations, especially at the hip" (Long, 1971). During development of these additional bony areas, active inflammation and swelling may occur and resembles an acute arthritic attack. The condition most often occurs within the first few months after injury, though in some cases it may occur many years later. It is a self-limiting condition, usually

progressing over several months to its ultimate anatomic limit.

Formations of these bony processes always occurs below the level of the spinal lesion and usually is limited to a specific muscle group. It occurs most frequently at a level between the upper pelvis and the knee. However, it may also occur in the paraspinal muscles. There is no known treatment of this complication. However, surgery is sometimes performed when the restriction of motion causes severe limitation of activity, generally at the hip joint (Long, 1971).

E. Spasticity:

Spasticity occurs when there is an increase in the stretch reflex. It has been found that a moderate state of spasticity is not harmful and in fact may be somewhat advantageous, as it aids in the transfer activities of the spinal cord injured individual and can contribute to a lessening of the amount of osteporosis. However, the occurence of severe spasticity intereferes with activities of daily living, causes discomfort and can further the complications of decubiti and contracture formation (Carroll, 1962; U. of Colorado, 1962). Severe spasticity is found in relatively few spinal cord injured cases, however, when present, it is an urgent problem. The management of severe spasticity is generally unsatisfactory, though various techniques and therapies have been utilized to lessen the negative effects of this condition, i.e. drugs, stretching, electrical stimulation, braces, tranquilizers, peripeheral operations (neurotomies, myotomies, tenotomies), local injections and rhizotomies (Kapla , 1966).

The amount of spasticity incurred by an individual varies from day to day and hour to hour. It appears to be related to heat, cold, emotional attitudes, the status of the urinary and gastrointestinal tract, decubiti and other irritant or painful stimuli (Kaplan, 1966). The cold, sitting or remaining still for long periods of time and pain or any stress (emotional, gastrointestinal, genitiurinary), all tend to increase the amount of spasticity, while heat, standing, walking or moving diminishes spasticity (Kaplan, 1966). A review of the literature indicates the incidence of spasticity varies from 11% to 65% of the spinal cord injured population (Zankel, et al., 1954; Hastings, 1959; Kaplan, 1966). The first occurence of spasticity tends to be within one year after a spinal cord injury (if the causative factor is trauma and/or the lesion is nonprogressive), but often occurs at a later stage of the lesion is progressive in nature. Spasticity can cause the following problems: awakening one from sleep; being "shot back" in a chair; increasing backache; producing discomfort when wearing braces; increasing difficulty in ambulation and proper transfer (Kaplan, 1966).

The area in which spasticity occurs differs for paraplegics and quadriplegics. Among paraplegics, spasticity is present in either the flexors and/or the extensors of the lower extremities while among quadriplegics spasticity can be found in the upper extensors and/or flexors as well (Hastings, 1959; VA Administration, 1948).

F. Contractures:

Contractures result primarily from joints remaining in one position for a long enough period of time to permit the shortening of supporting structures (i.e. ligaments, muscles, joint capsules). Contractures produce a distortion of the peripheral joints, especially those of the knees, hips, elbows and fingers. Casual factors associated with contractures include: remaining in bed and/or wheelchair too long; the bed being too short; and non-use of braces. Most of the patients with new or worsening contractures were not standing nor walking during the interim period (Kaplan, 1966).

This complication can be alleviated through motion, either in the form of passive or active motion. Passive motion therapy should be started early (i.e. the day after injury), and conducted twice daily with full passive range of motion for each joint. However, if this therapy and the additional techniques of gradual stretching bracing or splinting are not successful, surgical procedures can be implemented as a last resort (Rusk, 1964).

Contractures complicate the management of approximately 20% of the quadriplegics. And overall, there is a greater incidence of flexion contracture than extension contracture (Rusk, 1964; Kaplan et al., 1966).

G. Dysfunctioning of the Excretory System:

Spinal cord injury often results in a loss of normal bladder functioning. Comarr (1956) notes that approximately one-half of patients with complete lesions and approximately three-quarters of those with incomplete lesions develop reflex or neurogenic bladder.

The remaining 25 to 50% may require several operative procedures prior to satisfactory bladder control. The lack of voluntary bowel control is found in all patients with complete lesions.

Procedures have been developed to aid the spinal cord injured individual with the effective management of this health problem. And often the psychological hang-ups of improper excretory functioning are more devastating than the physiological complications.

H. Dysfunctioning of the Reproductive System:

"The physician (nor anyone else) cannot from onset of injury forecast the future return of sexual functioning for any one patient" (Comarr et al., 1972). Although some knowledge of the physical extent of the injury is generally an indicator of the extent of future sexual functioning, often times psychological and sociological factors rather than physiological factors influence one's potential sexual functioning. Furthermore, the absence of pleasurable sensations is more distressing than the mental or physical sense of inadequacy (Kaplan, 1966).

An injury to the spinal cord can result in some physiological limitations. These limitations are directly related to the: (1) neurological level of the lesion; (2) completness or incompleteness of the lesion; (3) presence of reflex activity through the sacral segments and (4) nature of sacral sparing. If the lesion is complete, the cord is transected, and there is no feeling below the level of transection, there is a possibility of erection though very little chance of ejaculation. With an incomplete lesion, there still may be partial feeling beneath the level of injury, and both erection

and ejaculation are possible (Sex and Paraplegia, 1972). There should be a physical examination to ascertain the degree of muscle tone in the rectum (i.e. external sphincter; pin-prick sensations in the penile skin, scrotal skin and saddle area). The patient must be able to voluntarily contract and relax his/her sphincter in order to participate in the more typical sexual behaviors. If the male patient has pin-prick sensation intact in the penile skin, scrotal skin and saddle area, it is a good indication of normal bladder, bowel and sex when the rectal sphincter is also functioning normally.

While studies indicate that up to 70% of all spinal cord patients are capable of having some degree of sexual activity, many do not indulge in any type of sex and have a poor attitude, believing a cord-injured person should not engage in this type of activity (Zeitlin et al., 1957). However, recently the spinal cord injured have begun to demand to know what their sexual limitations are and how to realize their full sexual capacity (Ryan, 1961).

According to Rusk (1967), a male should be able to have erections and ejaculate if the sacral cord, cauda equina and thoracolumbar sympathetic outflow are intact. Since, these two centers are far apart, a lesion may affect one and leave the other intact, which means that many times the injured man can have erections but not ejaculate.

It is uncertain whether or not the fertility of spinal cord injured females is significantly lower than that of nondisabled females. It is quite evident though that disabled women of child-

bearing years can and do become pregnant, and have full-term healthy babies (see Bradley et al., 1957; Comarr, 1966; Desmond, 1970; Goller, 1970; Guttman et al., 1965; Hardy and Warrell, 1965; Jackson, 1964; Mulla, 1957; Oppenhimer, 1971; Robertson and Guttman, 1963; Rossier, et al., 1969; Saunders, 1968).

There is also considerable debate over the extent of hormonal imbalance among cord-injured women. Comar (1966) and Cooper and Hoen (1952) believe no major imbalance of sex hormones exists in spinal cord injured women. To substantiate their claim, they cite the fact that pre-menopausal women invariably resume menstration following injury, often within a few months. Furthermore, Talbot (1955) reports that in cases of preadolescent lesions, menarche is not delayed at all. However, Bailey, Checkles and Johnson (1968) discovered numerous cases of women with high level cord lesions having alterations in their menstrual cycles. They cite the supportive observations of Durkan (see Bailey, Checkle and Johnson study) who found "...consistently low gonadotropin levels associated with vaginal cytology indicative of low estrogen levels."

The normal female counterpart of ejaculation is a biplasic motor response. The first phase is dependent upon sympathetic fibers remaining intact in the lower thoracic and upper lumber cord segments (Bors, Comarr, 1969), and consists of contractions of smooth muscle of fallopian tubes, uterus, and paraurethral glands of Skene. Phase Two, the contraction of striated muscles of the pelvic floor, perineum and anal sphincter, is activated by fibers in the S2-4 cord segments. Thus, the nerve fibers involved in

activation of the biplasic motor responses are located in the lower cord segments. The sensory component of the muscles involved in the biplasic motor response are the basis, at least in part, for the perception of orgasm. The nature and frequency of these reflexes in cord-injured women is unknown. Jackson (1972) believes few experience orgasm, although feigning a response is fairly common. Guttman (1969) claims that orgasm during intercourse is absent in women with complete lesions. Furthermore since the nerves below a cord lesion do not function normally, an injury in the cervical and upper thoraic region as well as in the lower thoracic and upper lumbar cord area would probably result in dysfunctioning of the biplasic motor response and thereby adversely affect orgasm.

TABLE 2-2

LIFE FUNCTION TABLE - SPINAL CORD INJURY: HEALTH

Statement of Problem	Dynamics	Source
Mild sweating to massive diaphoresis (excessive perspiration)	Result of autonomic hyper-reflexia SCI individual can manifest varying degrees of this problem throughout his/her life.	Cardus, et. al., 1969
Severe hypertension	One of the more severe responses to autonomic hyper-reflexia	Cardus, et. al., 1969
Headache	Symptom of autonomic hyper-reflexia	Cardus, et. al., 1969
Calculi in: 1. kidneys 2. urethera 3. bladder	Incidence figures range from 1.2-25.8% with the average being 10-11%	Bors & Comarr, 1971
Infections in excretory system organs: urinary infections	Common problem, often caused by indwelling catheters	Long, 1971
Neurogenic (reflex) bladder	Occurs in 50% of the patients with complete lesions; 75% of the patients with incomplete lesions	Comarr, 1956
Loss of bladder control		Guttmann, 1971
Heterotopic bone formation	Self-limiting condition often occurs within the first few months after injury	Long, 1971
Lack of voluntary bowel control	Occurs in all patients with complete lesions	Guttmann, 1971
Physical inability for sexual relations	30% of spinal cord injured male patients	Zeithlin, et. al., 1957

Table 2-2 cont'd.

Statement of Problem	Dynamics	Source
Tissue breakdown	Results from interruption of nutritional supply due to complete immobility	Felton, Perkins & Lewin, n.d.
Pulmonary complications	Among quadriplegics who have lost the ability to cough and clear bronchial secretions	Felton, Perkins & Lewin, n.d.
Osteoporosis	The longer the period of immobilization, the greater the osteoporosis	Felton, Perkins & Lewin, n.d.
Bone atrophy	Due to osteoporosis	Felton, Perkins & Lewin, n.d.
Pathological fractures	Due to osteoporosis	Felton, Perkins & Lewin, n.d.
Heterotrophic calcification	Due to osteoporosis	Felton, Perkins & Lewin, n.d.
Spinal shock	Onset of injury and for a period of 2 to 8 weeks; ends with gradual return of reflex activity	Guttmann, 1973; Hardy & Rossier, 1974; Ruge, 1969; O'Connor & Leitner, 1971
Decubitus ulcers	Due to prolonged immobility can be alleviated through proper health regime	Malament, Dunn, & Davis, 1975; Rosalie, et. al., 1975; McKenzie & Rogers, 1973; Guttmann, 1973; Hardy & Rossier, 1974; Pearman & England, 1973; Ruge, 1969; Austin, 1969; Rothman & Simeone, 1975
Large intake of water for proper regulation of bowels	Aspect of proper bowel control management	

Table 2-2 cont'd.

Statement of Problem	Dynamics	Source
Erections but not ejaculations	Depends upon the extent to which sacral cord, cause equina and thoracolumbar sympathetic outflow are severed.	Rusk, 1967
Alterations in menstrual cycle	Women with high level cord lesions	Bailey, Checkles & Johnson, 1968
Low estrogen levels	Spinal cord injured females	Durkan
Resicoureteral reflex		O'Connor & Leitner, 1971
Kidney inflammation	Usually due to kidney infection	Gutmann, 1973
Shortened life expectancy		Gutmann, 1973
Renal failure	Primary cause of death	Gutmann, 1973
Impaired body temperature control	During early posttrauma period	Gutmann, 1973
Phantom pains and sensations	Usually spontaneous; subside 6-8 months after onset of injury	Kaplan, et. al., 1966; Bors, 1959
Disturbances to the autonomic nervous system	Early stages of injury, temporary situation	Cardus, et. al., 1969
Significant weight loss which is not regained	Occurring from one month to one year following SCI trauma	Ruge, 1969
Muscle wasting	Due to complete immobility	Felton, Perkins & Lewin, n.d.
Impaired circulation	Due to complete immobility	Felton, Perkins & Lewin, n.d.

Table 2-2 cont'd.

Statement of Problem	Dynamics	Source
Trauma to the spinal cord 1. cervical 2. thoracic 3. sacral 4. lumbar 5. complete 6. incomplete	Possible sites and degrees of injury to the spinal cord	Guttmann, 1973

Mobility:

The extent to which a spinal cord injured patient's mobility is limited is related to the location of the lesion and whether it is complete or incomplete. The lower the level of injury, the greater the amounts of muscle power available to the patient for his rehabilitation (Long, 1971). Thus there is a decrease in the extent of mobility impairments as the site of injury descends downward. For example, an individual injured at the C5 level sustains more mobility limitations than an individual incurring injury at the C7 level. Since certain functional groups of muscles are activated at particular levels of the spinal cord, it is possible to categorize the potential performance of patients injured at and between these levels. Table 2-3 identifies the major muscles usable when injury occurs at various levels.

TABLE 2-3

CRITICAL LEVELS OF CORD FUNCTION

Level:	Major Muscles Added:
C5	diaphragm, midcervical extensors and flexors
C6	deltoid, biceps
C7	radial wrist extensors, serratus, latissimus, pectorals
C8	triceps, long finger flexors and extensors, wrist flexors and extensors
T1	hand intrinsics, ulnar wrist
T12	full abdominal innervation, full intercostal innervation
L4	hip flexors, knee extensors

Source: Long, 1971, pg. 36.

TABLE 2-4

LIFE FUNCTION TABLE - SPINAL CORD INJURY: MOBILITY

Statement of Problem	Dynamics	Source
Moderate spasticity	Incidence figures range from 11-63%; can be somewhat advantageous	Zankel, et. al.; Hastings, 1959
Severe spasticity	Found among relatively few spinal cord injured patients; however a major problem when it occurs	Carroll, 1962
Contractures	20% of quadriplegics; greater incidence of flexion contracture than extension contracture	Rusk, 1964; Kaplan, et. al., 1966
Need for wheelchair accessibility to public & private buildings	Wheelchair bound	Cass, 1974; Stock & Cole, 1975
Need for wheelchair accessibility for public transportation	Wheelchair bound	Cass, 1974
Need for barrier-free environment	Wheelchair bound	Prast, 1974
Difficulties in navigation through the environment: curbs, steps	Wheelchair bound	Prast, 1974
Need for adequate toilet facilities in public and private buildings	Wheelchair bound	Duckworth, 1975
Attendant aid for toilet transfer; attendant aid for car transfer	Wheelchair bound	Duckworth, 1975
Need for modification of private transportation, i.e., hand controls	Wheelchair bound	Ebel

TABLE 2-4 cont'd.

Statement of Problem	Dynamics	Source
Need for wheelchair accessibility in living environment	Wheelchair bound	Stock & Cole, 1975
Ability to perform cooking & task	Paraplegics	Range, 1966
Cannot travel on air transport	Quadriplegics & paraplegics	
Assistance needed to transfer to & from bed	C-6 level injury	Long, 1971
Propel wheelchair with hand rim projections	C-6 level injury	Long, 1971
Weak wrist extension	C-7 level injury	Long, 1971
Limited digital extension	C-7 level injury	Long, 1971
Assistance needed to transfer to commode chair	C-7 level injury	Long, 1971
Varying degrees of upper back, abdominal innervation & intercostal innervation	T-1 & T8 level injury	Long, 1971
Drive car with hand controls	C-7 - T8 level injury	Long, 1971
Can stand with the use of long leg braces however cannot walk	T-1 & T-8 level injury	Long, 1971
Independent in ADL activities at wheelchair level	T-1 & T-8 level of injury	Long, 1971
Attendant aid for dressing	C-6 level of injury	Runge, 1966
Attendant aid for fastening clothing	C-7 level of injury	Runge, 1966
Attendant aid in grooming		Runge, 1966
Unable to engage in simple food preparation		Runge, 1966

TABLE 2-4 cont'd.

Statement of Problem	Dynamics	Source
Need for use of adaptive equipment for self-care activities	Quadriplegics	Runge, 1966
Ability for wirtten communication limited	Need for adaptive devices quardriplegics	Long, 1971
Ability to transfer self from bed to wheelchair	C-7 level of injury & below	Long, 1971
Ability to transfer self from wheelchair to car	C-7 level of injury & below	Long, 1971
Paralysis of all limbs except for limited neck motion	C-4 level injury	Long, 1971
Incapable of voluntary functioning in arms, trunk or lower extremities	C-4 level injury	Long, 1971
Partial shoulder movement	C-5 level injury	Long, 1971
Weak elbow flexion	C-5 level injury	Long, 1971
Manipulate electric wheelchair with mouth stick	C-5 level injury	Long, 1971
Limited self-care using arm supports & externally powered hand splints	C-5 level injury	Long, 1971
Very poor grasping ability	C-6 & C-7 level injury	Long, 1971
Graded control of the wrist, with gravity performing the flexion movements	C-6 level injury	Long, 1971

A study conducted by Runge (1966) supports the contention that the number of activities a spinal cord injured individual can perform in directly related to the level at which the lesion occurred. Data was collected on the performance of self-care activities by 48 spinal cord injured individuals after discharge from a hospital. (Please note: individuals had cervical level injuries). The areas of self-care activities examined were: feeding, grooming, writing, typing, fastening clothing, dressing upper extremities, dressing total, driving and light household skills (i.e. simple food preparation). The following table summarizes the findings:

TABLE 2-5

Level of Injury:	Self-care activities: (50% or more of the individuals presently independent in:)
C5	typing, feeding, writing
C6A	feeding, writing, typing, grooming
C6B	grooming, feeding, writing, typing, fasten clothing, dressing upper
C7A	feeding, grooming, writing, dressing upper
C7B	feeding, grooming, writing, fasten clothing, dressing upper, driving (43% at discharge & 43% presently) dressing total (14% at discharge & 43% presently) Household (0% at discharge & 43% presently)
C8	feeding, writing, grooming, fasten clothing, dressing upper, household, dressing total, driving (66% presently)

Source: Runge (1966)

C8 patients did not have to learn substitution patterns or tricks to do most of the self-care activities. These individuals should be independent in all of the nine self-care activities unless there are specific problems (i.e. severe spasticity). C8 spinal cord injured persons may not even need professional training to gain this self-care independence (Runge, 1966).

A direct relationship was found between continued use of adaptive equipment and the ability to perform the same number of self-care activities after hospital discharge. Fifty-two percent of the patients using less equipment after their discharge performed a decreased number of self-care activities. Only 14% of those patients using the same amoung of adaptive equipment after hospital discharge decreased in the number of self-care activities performed (Runge, 1966).

Even the most functionally limited spinal cord injured individual can achieve some form of mobility functioning with the aid of adaptive devices; and a concerted effort should be made by the patient and rehabilitators to achieve specific mobility goals. Guttman (1973) has compiled a list of specific mobility goals for the various levels of spinal cord injury. These goals reflect the maximum in physical functioning according to the level of injury.

Spinal Cord Level	Key Muscle Control	Functional Goals (Mobility Goals)
C1-C3		-injuries to these levels are usually fatal due to total respiratory paralysis.
C4		-total scope of movement: limited neck motion -incapable of voluntary function in the arms, trunk or lower extremities

Spinal Cord Level	Key Muscle Control	Functional Goals (Mobility Goals)
C4 cont.		-upper extremity function possible only with use of externally powered devices.
C5	neck, upper trapezius	-good neck motion; partial shoulder movement; weak elbow flexion. -manipulate electric wheelchair with mouthstick. -limited self-care using arm supports & externally powered hand splints: feeding, some grooming activities; help somewhat with upper extremity dressing. -help apply bracing; turn pages; use the electric typewriter.
C6	shoulder muscles, elbow flexors	-shoulder motion, elbow flexion; graded control of the wrist, with gr[illegible] performing the flexion movements; [illegible]st extension can be harnessed through special "tenodesis" splints to drive the fingers into flexion; very poor grasping ability. -self-care activities: self feeding with externally powered hand splints or clip-on equipment; light hygiene (make-up, brushing teeth); dress upper trunk. transfers: to and from bed with assistance; car transfers possible. -turn self in bed with overhead arm slings. -propel wheelchair with hand rim projections.
C7	wrist extensors, supinators	-shoulder motion; elbow flexion; weak wrist extension; very poor grasping ability; elbow extention; limited digital extension. -transfer to and from bed and auto without assistance. -transfer to commode chair with assistance. -propel chair with hand rim projections. -self-feeding, hygiene (shaving and grooming hair) dressing, writing and skin inspection using wrist driven flexor hinge hand splints where prehension is required. -drive auto with hand controls.

Spinal Cord Level	Key Muscle Control	Functional Goals (Mobility Goals)
C8	elbow extensors, weak hand	-transfer independence to and from bed, car, toilet. -propel wheelchair without hand rim projections. -wheelchair in and out of car. -independent in all self-care activities without hand splints or adapted equipment except catheter. -can participate in household duties from wheelchair.
T1-T8	hand muscles	-full innervation of upper extremities. -muscles and varying degrees of upper back, abdominal and intercostal innervation. -may be braced (long leg braces) for standing, but should not be expected to walk functionally. -independent at the wheelchair level; can dress and feed himself/herself; tend to his/her toilet; transfer to and from wheelchair. -including to and from tub; manipulate wheelchair up and down curb; transfer self in and out of car; drive car with hand controls.

Source: L. Guttman, 1973

Social Attitudinal:

The quadriplegic, though mentally independent, has lost all of his/her physical independence (Siller, 1969; Maxwell, 1971). Kahn (1969) notes that the person with a spinal cord injury often feels a loss of control over his/her life. Fears develop concerning the individual's health status, appearance, loss of status in the community and within his/her family. Additionally, there is a fear of loss of sexual functioning. Some individuals cannot even cough or sneeze due to lose of muscular contraction within the chest. This feeling of dependence remains in his/her life, and often the "most damning consequence" of

spinal cord injury is the constant emotional destruction resulting from dependence on others (Maxwell, 1971). Furthermore, some patients become dependent upon others for all their emotional and bodily needs (Mueller, 1974). Many individuals feel the loss of prestige due to increased dependency (Nagler, 1950). Furthermore, it is difficult for a nondisabled individual to comprehend "the deep fright a paralyzed person must feel over the possibility of antagonizing anyone on whom he must depend for the simplest functions" (Mann, Godfrey, and Dowd, 1973).

The individual not only becomes dependent, but may adopt a passive behavior. Passivity is defined as the: "mode of adaptation to one's environment that involves going back from it." A passive person waits for things to come to him/her rather than initiating action. That is, the individual responds to situations as they develop. This mechanism can be useful to some extent, in that it protects the spinal cord injured individual from embarassing situations. However, it may also result in the inability to accept responsibility for one's own rehabilitation (Siller, 1969). "The manifestation of withdrawal and isolation mechanisms minimizes the focus on self, but creates a lack of spontaneity and initiative" (Seymour, 1955).

Depression is commonly manifest by spinal cord injured individuals. A study of 109 patients concluded that 45% showed some manifestations of episodic or prolonged depression (Thom et al., 1946). Wittkoer (1954), after observing 70 patients noted that all exhibited deep depression during the early months of the disability, with some hoping for death

(though none attempted suicide). Weiss and Bors (1948) interviewed 210 paraplegic patients. They were found to suffer from despair and bitterness due to infection, pain, prolonged illness and continued incapacitation (i.e. paralysis of extremities, loss of sexual functioning, loss of control of bladder and bowel functioning) which can accompany injury to the spine. Nagler (1950) found that the spinal cord injured experienced immediate depression due to the realization of the severity of their illness. However, this depression was only temporary. Of a total of 700 patients interviewed and/or observed only 6 patients remained depressed for an extended period of time.

Clinically, anxiety and depression are the primary reactions to physical traumatization and usually are observed readily. Depression should be considered an appropriate response to traumatic spinal cord injury. "A person should be depressed because something significant has happened, and not to respond as such is denial." It is essentially denial that one's life will change substantially in important respects (Siller, 1969). According to Meuller (1950, 1974), situational depression and dependency are natural. In fact, if the spinal cord injured individual does not go through this depression, many times he is denying the injury and this could result in even more severe psychological problems. Fundamental to an understanding of any emotional reaction to a physical injury is the effect it has on an individual's body image. Each individual has an unconscious image of what his/her body looks like from the outside, and this image which is perceived by the ego, is constantly reinforced by both central and peripheral nervous system

stimuli. An abrupt change in peripheral nervous system stimuli, due to injury and/or mutilation, does not produce an immediate or automatic change in the ego's image of the body. "The ego is only slowly able to alter its concept of what the changed body looks like and that occurs only after a good deal of time and mental effort" (Ruge, 1969).

Spinal cord trauma is a fundamental threat to the body image. Therefore, the emotional reactions to such an injury tend to be defensive measures aimed at preservation of the old image and denial of the changes in body functioning (Starret and Grayson, 1961).

During the initial recovery period, the majority of paraplegics believe, full recovery is possible. The paraplegic can see his/her limbs with no apparent injury and believes he/she can move them and will eventually walk again. Sensory changes (i.e. sensation of phantom limbs) are interpreted as signs of returning function. Often specific rehabilitation treatments and surgery are rejected due to this type of wishful thinking (Bors, 1951).

Litin (1957) believes a crucial emotional climax comes with the realization of a changed body and therefore changed capacities for living. The psychologically disturbed reactions are caused by the patients efforts to master painful feelings that these realizations arouse in him.

> "...in working with disabled individauls, one is frequently impressed not with their depression, as one would imagine, but with their complete inability to understand what the disability is all about...There is a veritable struggle that takes place... They cannot find a place in their body image for deformity"

(Mueller, 1962). However, the body image must be reorganized to incorporate the deformed absent or useless body part within the individuals image of himself (Grayson, 1952). Grayson (1952) attributes symptoms such as depersonalization, feelings of unreality, and resistance to the use of orthopedic appliances to a defense the ego unconsciously sets up to maintain its integrity in the face of body image disturbances.

A behavior which may accompany or follow the comprehension of the extent of lost body functioning is "mourning behavior." The loss of a body part or of an important function can activate mourning reactions. Working through the myriad of meanings that a part of function has for the person cannot be hurried, nor should it be, for the reaction of grief is adaptive in that it prevents the complete breakdown of the ego (Siller, 1969). According to Siller (1969), one can finally accept the loss when the reconstituted self is oriented toward self-approval and begins to rely on protective mechanisms that are essentially nonpathological.

Often the perception of functional or physical limitations precipitates psychological problems. For example, some spinal cord injured individuals feel frustration, shame, embarrassment and inferiority due to loss of bladder and bowel control (Mueller, 1965; Thom et al., 1946; Weiss and Bots, 1948; Bors, 1955; Comarr, 1956). Numerous studies dealing with the sexual functioning of paraplegics reflect problems in the social-attitudinal area (Talbot, 1949, 1955; Bors, 1948; Munro, Throne and Pauli, 1948). Physically caused impotency

in spinal cord injured males results in feelings of inadequacy and the relinquishing of all aspects of "manly behavior" (i.e. independence, authority and responsibility) (Mueller, 1962). The loss of sexual function, excretory functions and physical abilities results in low self-esteem (O'Connor and Leitner, 1971). Furthermore, false hopes may exist and a shock effect is present due to the loss of sexual functions (Nagler, 1950). Often times changes in excretory functions result in guilt, shame and anxiety. Decubiti ulcers are related to cultural ideas of uncleanliness, contagion and veneral disease (Garett, 1952; Siller, 1969).

> The work of my research group clearly shows that sores and oozing ulcerous type conditions elicit some of the strongest aversion reactions... We, as well as others, have made the connection between surface sores and imputations of dirtiness, contagion and veneral disease (Siller, 1969).

To effectively treat a spinal cord injured patient, one must take into account the premorbid personality and certain demographic characteristics: sex, age of onset, cultural background and educational background. The individual's pretraumatic personality, social experiences and family constellation patterns play an important part in determining the response to a treatment program (Litman, 1962). This leads to the view held by several authors that the emotional reactions of a spinal cord injured individual are, to some extent idiosyncratic (Bors, 1959; Siller, 1969; Garrett, 1952). Nagler (1950) identifies seven distinct psychological types of reactions to spinal cord injury:

1. The individual mainfests anxiety and reactive depression. No attempt is made to adjust to the situation. However, there is a good prognosis due to the fact that these patients recover from these depressed feelings.
2. A paranoid schizophrenic reaction. It is believed that this reaction is not caused by the trauma per se but more probably is rooted in an early childhood disturbance (Bors, 1959). The spinal cord injury enhances the pretraumatic personality.
3. Total indifference to the situation. Usually less evident among spinal cord injured with high intelligence.
4. Psychopathic reaction manifesting the following behaviors: hostility, abusive language, resentfulness, hate and lack of emotional restraint.
5. Patients are extremely dependent and emotionally unstable. There is an attempt to avoid and escape from the reality of the injury. Often times, these individuals have a problem with drug addiction.
6. These patients, especially quadriplegics, have difficulty channeling aggressive drives into productive accomplishments. Although there is a continous effort to avoid anti-social aggressive behavior patterns, emotional explosiveness is often found.
7. Positive attitudes towards their disability and themselves: acceptance of the disability, good insight into their limitations and potentialities, and constructive future plans.

It has been hypothesized that what is observed psychologically in the spinal cord injured patient, is an exaggerated premorbid personality. "Where the person has always been mature and well adjusted he will react...in just that manner. Where the person has always been infantile and poorly adjusted there will be just that type of reaction to the disability" (Garret, 1952). According to Comarr (1956), the pretraumatic personality continues to set the pattern for postraumatic goals and behaviors. In general, the more intelligent patients with a strong desire to achieve adjustment develop the "so called normal reaction":

> This is the "ideal" we desire to accomplish. These men have made the best of it; they know their limitations and potentialities and have constructive plans. This does not mean that they are free from problems and anxieties. However, they have succeeded in sublimating their sexual drives into channels for constructive work and in compensating their inferiorities by gaining prestige and recognition in endeavors that they can master (Nagler, 1950).

The "indifferent group", patients with lower intelligence and little drive to achieve or adjust to their lives, often adopt an attitude of indifference, passivity and complete hopelessness. They are content to just let the other fellow worry and show no signs of overt aggression nor drive either negative or positive (Nagler, 1950).

However, while no specific personality type has been discovered for either paraplegics or quadriplegics, there appear to be certain common pre-morbid personality characteristics in all spinal cord

injured persons. It has been found that young male spinal cord injured patients (aged 15-40), prior to the onset of the disability, earned their living in some active or unskilled vocation (Wittkower, 1955). Many have passive-aggressive characteristics with a strong tendency to express their aggressive side directly. The most prevalent emotional pattern is adolescent egocentricity, with uncontrolled infantile affective behavior. Prior to the onset of injury, many spinal cord injured individuals were people of action rather than contemplation, with few inner emotional resources for gratification. These individuals are found to have poor personality controls, limited channels for emotional resources and punitive consciences. There is a tendency toward lack of good human identification or empathy. They also lack the capacity for fantasy as a means of discharging their impulses. Tension is relieved through physical action (Mueller, 1950; Garret, 1952; Siller, 1969; Ruge, 1969). According to Ruge (1969), "...those who are least well endowed to meet the massive challenge imposed by a spinal cord injury are frequently the very people who incur it."

After the onset of a spinal cord injury, an individual never resumes his former life or his former personality patterns and adaptations unchanged. The sudden, traumatic nature of the disablement necessitates a great deal of personal adjustment (Litman, 1962). The injury limits the discharge of aggressive energy, imposes a severe restriction of activity and requires the patient to rely largely upon verbal channels for relating to others and expressing feelings. Intellectual interests and fantasy become more important.

However, there are severe problems in the area of self-esteem (i.e. the injury is a threat to one's adult expectations), social and vocational roles and sexual identity and functioning. A number of negative emotional feelings are encountered--feelings of helplessness, dependency, hopelessness, negativism and self-destructiveness. Mueller's (1950) findings indicate that spinal cord injured patients show a tendency toward ambivalence, indecision, passive submission behavior, unrealistic thinking and immature emotional expression. In general, a wide variety of discrete psychoneurotic syndromes are displayed: anxiety, depression (prolonged and acute), hysteria, paranoia, and somatization and hostility toward the dependent role; as well as incapacitating or interfering emotional reaction states: hostility dependence, combative negativism and helplessness, apathy and resentment (Cath et al., 1957; Fordyce, 1964; Guttman, 1946; Kennedy, 1946; Labhardt, 1962; Mueller, 1962; Nagler, 1950). The development of the above negative tendencies are viewed as attempts to cope with threats to the individaul's self-esteem and adult expectations (Nemiah, 1957).

However, Shontz (1962) lists a number of negative reactions which may adversely affect rehabilitation efforts: gross confusion and uncertainty, panic, nonspecific anxiety, fear of loss of sanity, fear of death or dying, fear of becoming a physical, emotional and financial burden, feeling different from others, shame upon exposure, vulnerability to rejection, emotional or physical attack, feelings of self-punishment, suicidal thoughts and death wishes, uncontrollable

hostility, rejection by friends, family and society, feelings of being at the mercy of a cruel and unjust fate, feeling that adult status is completely lost and worry over availability of physical assistance (Shontz, 1962). Furthermore, Shontz (1963) lists a number of psychological reactions which may aid or at least not affect one way or the other (neutralizing reactions) the rehabilitation process. Neutralizing reactions include: intellectual denial of illness, withdrawal from emotional reactivity, conversion of unpleasant affect, relief from responsibility of life, decentralizing the disability in relation to total personality, feeling of having paid one's debt for past sins, exploiting others. Positive reactions include: fantasizing future success, thinking of the disability as a new chance to correct old errors, full appreciation of life by almost dying, faith and hope for a miracle cure, feeling of interpersonal worth derived from the care of others, feeling of being selected for spiriaual trials of suffering, developing good values from non-physical aspects of existence.

TABLE 2-6

LIFE FUNCTION TABLE - SPINAL CORD INJURED: SOCIAL ATTITUDINAL

Statement of Problem	Dynamics	Source
Loss of control over his/her life		Kahn, 1969
Loss of physical independence	Especially severe among quadreplegics	Siller, 1969; Maxwell, 1971
Fear of loss of status within family	Common reaction of spinal cord injured	Shontz, 1962
Fear of loss of sexual functioning	Common reaction of spinal cord injured	Talbot, 1949; 1955; Bors, 1948; Munvo, Thorne & Pauli, 1948
Loss of prestige	Common reaction of spinal cord injured	Nagler, 1950
Fright over possibility of antagonizing others	Common reaction of spinal cord injured	Mann, Godfry & David, 1973
Adoption of passive behavior	Common reaction of spinal cord injured	Siller, 1969
Withdrawal	Common reaction of spinal cord injured	Seymour, 1955
Depression	Common occurence; Thom, et. al., 1946 - 45% of sample exhibited episodic or prolonged depression. Other studies indicate depressed state is temporary & necessary for full recovery	Weiss & Bors, 1948; Nagler, 1950; Wittkoer, 1954; Thom, et. al., 1946; Siller, 1969
Anxiety		Weiss & Bors, 1948
Denial of injury	Block to rehabilitation	Bors, 1951; Mueller, 1962
Body image threatened		Starret & Grayson, 1961

TABLE 2-6 cont'd.

Statement of Problem	Dynamics	Source
Wishful thinking injury only temporary	Especially among paraplegics	Bors, 1951; Mueller, 1962
Reorganization of body image necessary		Ruge, 1969
Depersonalization		Grayson, 1952
Feelings of unreality		Grayson, 1952
"Mourning behavior"		Siller, 1969
Frustration		Mueller, 1950; Comarr, 1956
Shame		Thom, et. al., 1946; Weiss & Bors, 1948; Bors, 1955
Embarrassment		Bors, 1955
Inferiority		Mueller, 1950
Passive-aggressive personality	Premorbid personality characteristics males 15-40	Wittkower, 1955
Adolescent egocentricity	Premorbid personality characteristics males 15-40	Wittkower, 1955
Few inner emotional resources for gratification	Premorbid personality characteristics males 15-40	Nagler, 1950
Punitive consciences		Nagler, 1950
Fantasy	Becomes more improved after the injury	Litman, 1962
Self-destructiveness	Common during initial recovery period	
Hopelessness		
Apathy		
Resentment		

TABLE 2-6 cont'd.

Statement of Problem	Dynamics	Source
Sexual dysfunctioning	70% of spinal cord injured physiologically unable to engage in some form of sexual activity	Zeitliz et. al., 1957
Low self-esteem	Physical dysfunction	Mueller, 1930
Guilt		Shontz, 1962
Paranoid schizophrenic reaction to disability	Not caused by trauma per se but rooted in early childhood disturbance	Bors, 1959
Totally indifferent to situation		Nagler, 1950
Emotional instability		Nagler, 1950
Attempt to avoid & escape from reality of the injury		Nagler, 1950
Emotional explosiveness	Quadriplegics who have difficulty channeling aggressive drives	Nagler, 1950
Pretraumatic personality continues to set pattern for postraumatic goals & behaviors	Negative effect if individuals immature & poorly adjusted in society	Wittkower, 1955
Subjective feelings of impending doom	Symptom of autonomic hyperreflexia	Cardus, et. al., 1969
Gross confusion & uncertainty	Will adversely affect rehabilitation efforts	Shontz, 1962
Fear of loss of sanity	Will adversely affect rehabilitation efforts	Shontz, 1962
Fear of becoming burden on the family - physically, emotionally & financial		Shontz, 1962

TABLE 2-6 cont'd.

Statement of Problem	Dynamics	Source
Feeling of being different from others		Shontz, 1962
Suicidal thoughts		Shontz, 1962
Feeling of having paid debt for past sins		Shontz, 1962
Fantasizing future success		Shontz, 1962
Thinking of the disability as a new chance to correct old errors		Shontz, 1962

Communication

A spinal cord injured individual is not significantly impaired in the communication life function. Although the individual is potentially limited in the area of written communication, the ability to hear, to see and to speak is not affected by his/her disability. Furthermore, adaptive devices have been created to compensate for the loss of hand manipulation and thus the ability to write (i.e. specially equipped typewriters, voice activated tape recorders to be utilized as notetakers).

Cognitive-Intellectual

In most instances, even when there is extensive spinal cord injury, an individual does not suffer from brain damage (Felton, Perkins and Lewin, n.d.). The large number of spinal cord injured individuals enrolled in colleges and universitites, as well as employed in professional and white collar jobs, attest to the lack of cognitive-intellectual impairment (see section on: Functioning as a Member of the Labor Force, for further information). Furthermore, a group of individuals studied by Muller and Thompson (1950) were found to have a significantly higher intellectual capacity than the general population.

VI. Functioning as a Member of Society

The literature documents the spinal-cord injured's functioning as a member of the labor force, the community, and the family.

Functioning As a Member of the Labor Force

It would appear that the extent of the spinal cord injury has a significant impact on achieving employment. Long and Lawton (1955) related the level of spinal cord injury to expected work capabilities and found the following:

TABLE 2-7

Level at which lesion sustained	Work Activity
Quadriplegics (complete or partial)	
C-5	The individual, can use a telephone or typewriter.
C-6	The individual can utilize a greater variety of adaptive machinery and typing skill is usually more proficient. Possible use of monograph tools or addressing machinery.
C-7	Possible occupations: bookkeeping, mimeography, telephone services, typing or use of simple office machines. Full use of the hands. Some individuals are able to drive hand-controlled vehicles but usually require assistance in entering and exiting from the vehicle.
Paraplegics (complete or partial)	
T1 - T12	Individual has few vocational limitations in non-ambulatory and semi-ambulatory jobs away from home, usually wheelchair not needed.
L-4 lesion	The only limitation in potential job placement would be in jobs which require standing for extended periods of time. "Employment requiring constant rising from a chair or a great amount of elevation activities is not feasible.

Source: Long & Lawton (1955)

The Dvonch (1965) study notes that 75% of those quadriplegics sampled could drive to their place of employment. Thus, tending to indicate that the more self-sufficient quardiplegic is employable, while those quadriplegics, with more severe functional limitations, are not.

A study of 1,204 spinal cord injured persons, attending the Lyndhurst Lodge Hospital in Toronto, Ontario, reported that 51% were employed or attending school. However, the extent of the injury was found to affect employability and/or school attendance. The percentage of those individuals attending school or employed by extent of injury are listed below:

32% = complete quadriplegic
41% = partial quadriplegic
54% = complete parapletic
58% = partial paraplegia

The researchers concluded that the higher and more complete the spinal cord lesion, the more difficult it was to achieve reemployment. Furthermore, the benefits derived from higher educational attainment varied by level of spinal injury: (Geisler et al., 1966)

> "Between complete quadriplegics with primary school education and those with secondary education, the improvement with higher attainment was doubled. Further doubling occurred when specialized post-secondary school education was recorded. Though not as dramatic, the trend was much the same with partial quadripletics. For paraplegics, however, specialized post-education did not appear to matter to such an extent, although benefits of secondary educational attainment over primary were still quite marked."

Numerous studies have gathered data to delineate those factors which contribute to the employability of the spinal cord injured individuals. It has been found that the level of education is a significant contributor to achieving gainful employment. (Berns et al., 1957; Coble, 1950; Dvonch et al., 1965; Felton and Litman, 1965; Geisler, 1966; and Short, 1973). Felton and Litman (1965) report that the unemployed had only a small increase in additional education between the predisability and postdisability periods as compared with employed spinal cord injured individuals. Furthermore, Seigel (1966) notes: "Our experiences with quadriplegics possessing a college education indicate that quadriplegics who successfully complete a college program have excellent prospects for successful competitive employment." Therefore, educational attainment prior to injury and after onset of the injury are important contributory factors to employment (Geisler et al., 1961).

Other factors influencing employment were found to be age of onset of the injury, urban geographical location, high level of intelligence, positive motivation, sustained good health, ability to drive a car, good work record prior to the disability (Geisler et al., 1966; Walker, 1961; Dvonch et al., 1965).

Dvonch et al. (1965) conducted a study comparing the state of employment at the beginning and end of a three year period. All of the 55 subjects had been employed at some time during the three year period, only 30 were still employed at the end of the three years. A number of characteristics differentiated the employed from the non-employed:

1. "Those employed had a mean age that was 5.9 years younger than those who were no longer employed."
2. "Those employed constituted 73.9% of the total number of high school graduates."
3. Those employed showed a much more stable work history in their employment prior to, and after, onset of disability."

Geisler et al. (1966) conclude that younger patients are more employable than older patients due to the former's greater adaptability, strength and energy. However, no data is produced to support their contention. Hastings (1959) reports that health factors influence the employer's hiring practices. No patients with severe spasticity were employed, while between 39.8 and 68.6% without spasm were employed.

According to Trigano and Mitchell (1970), previous work experience facilitates employment opportunities outside the home. Dvonch et al. (1965) note that work stability prior to the onset of the injury is an important variable relating to successful vocational rehabilitation. Walker (1961) believes previous work experience to be a positive factor for reemployment at the same work site. Levenson and Green (1965) suggest that the chances for vocational success would be higher if the quadriplegic could return to their former employment and even greater chances of success if they could do their own job. However, Fleton and Litman (1965) after sampling 222 paraplegics found pre-injury occupation to have little relationship with post-injury employment. Vocational rehabilitation training did not significantly aid the disabled in finding employment. The majority of the individuals were trained for only a few specific occupations, with clerical jobs

leading the list. However, few of the respondents were working in the jobs they had trained for, and only 13% of the employed obtained jobs as the result of vocational rehabilitation services. The primary means of job obtainment were referrals by friends and self-employment. As of December, 1971, of a total of 303 spinal cord injury patients served by the Southwest Regional System for the Treatment of Spinal Cord Injury, 208 individuals were determined qualified to work. However, only 48.1% (100) were working or in training.

In general, the vocational outlook for quadriplegics is poor and only a small percentage of quadriplegics reach viable vocational goals (Bors, 1959; Shontz, 1962; Little et al., 1974). In 1955, only 8% of the quadriplegics interviewed were gainfully employed (Bors, 1955). Though recently, studies indicate some increase in the numbers of quadriplegics and paraplegics holding jobs. Occupations presently held by quadriplegics include: clerical, sales, personnel, professional, technical, and managerial positions (Kaplan, 1966; Bors, 1959; Walker, 1967; Rusk, 1963; Seifel, 1969).

Rusk (1963) conducted a follow-up study of 355 quadriplegics who attended the Institute of Physical Medicine and Rehabilitation between 1948 through 1960. There were 177 respondents; 69 (39%) were gainfully employed and 18 (10.2%) were attending college. The occupations of the 69 persons employed were classified as follows:

27.8% = professional positions
27.8% = managerial positions
24.1% = clerical positions
12.8% = sales
7.4% = skilled positions

Runge (1966) conducted a six month follow-up study of 48 individuals discharged from Ranchos Los Amigos Hospital in California. Twenty-nine percent of these individuals were either employed or attending school. Furthermore, except for the C-5 level injured, some individuals with lesions at all remaining levels were employed or in school.

TABLE 2-8

Total No. of Patients	Spinal Cord Injury Level	Patients Employed or in School According to Motor Levels.
5	C5	0
6	C6A	2
10	C6B	3
17	C7A	6
7	C7B	2
3	C8	1
48		

Source: Runge, 1966

Occupations for those employed included: receptionist, record store proprietor, stockbroker trainee, accountant, government worker, medical resident in pathology. Those enrolled in school attended: high school, two-year junior colleges, four-year colleges and universities.

A vocational rehabilitation study conducted in Canada found of a total of 343 quadriplegics only 34% were employed and 10% in school or training (Geisler et al., 1966). However, a study conducted by Kaplan (1966) indicated more positive success (though it should be noted that Kaplan's sample included paraplegics as well as quadriplegics). Of a total of 131 patients admitted to the Institute of Rehabilitation Medicine between 1962 and 1967: 44 (34%) were competitively employed, 3 (2%) were homemakers, 62 (47%) were in college or special training

(vocational) programs and 22 (17%) were unemployed at the time of the interview. The 44 employed individuals included: 36, working in regular places of employment; 5, performing all or part of their work at home and 3, operating small businesses. The 62 persons in educational programs included 59 in college and 3 in vocational training programs (Kaplan, 1966).

While quadriplgeics are employed in every major occuaptional category, the majority are in unsatisfactory homebound occupations (Walker, 1961; Siegel, 1969). Walker (1961) notes that "...the inabilities of patients to achieve appropriate goals do not represent failures by the patient, but by rehabilitation personnel who have not developed techniques and programs which tap patient resources. Patient failures cannot be justifies on the basis of rationalization that such severely disabled patients cannot work."

If there were effective rehabilitation services available, quadriplegics could be more successfully employed. A coordinated effort between agencies results in a higher success rate in placement of quadriplegics in work situations (Selwyn, 1964). Rusk (1963) believes a high percentage of young quadriplegics can return to work when there is an effort at intensive job and school placement services in a medical rehabilitation center.

Izard and Staton (1969)conducted a demonstration project with 25 quadriplegics labeled unfeasible for rehabilitation. These individuals were introduced to a sheltered environment with a well qualified professional staff and all the necessary medical services. Vocational

goals were emphasized, with the result that 52% could be placed in work or school. This compared with the the national average of approximately 10%. It was concluded that the following policies should be implemented for an effective program:

1. clients should be made as healthy as possible.
2. communal living should be considered, as this decreases the need for attendants.
3. individuals not interested in work should be weeded out early.
4. a variety of vocational goals should be made available.
5. emotional factors should be minimized.
6. recreational facilities should be made available.

In general the spinal cord injured are successful in occupations based upon intellectual and educational qualifications rather than manual abilities (and/or ambulatory abilities). Although a relatively small number of quadriplegics have been successful in occupations requiring some form of manual labor, there must be further and more extensive development of programs in these occupational areas. Especially since not all quadriplegics can qualify for white collar positions (Seigel, 1969).

A number of other problems exist for the spinal cord injured individual. These include:

1. lack of employment placement service at hospital (Ramsey, 1974).
2. lack of sufficient rehabilitation counseling for job placement (Schlenoff, 1975).
3. lack of recognition of limitation of work capabilities (Crew et al., 1975).

4. lack of placement in competitive employment (Gibson, 1975).
5. low pay, underemployment and lack of full employment (Patrick, 1975; Runge, 1966; Dvonch, et al., 1965, Berns et al., 1957; Kaplan, 1966; Kirkpatrick, 1967, Spangler, 1966; Walker, 1961).
6. immediate cessation of disability payments upon finding employment (Wise, 1974).
7. lack of good physical work accomodations (i.e. need for access to parking space close to the worksite, barrier-free navigation of wheelchair in work setting, toilet facilities readily accessible) (Walker, 1969).

Functioning as a Member of the Community

According to Cogswell (1968), upon returning home from the hospital, paraplegics have a marked reduction in the: (1) number of social contacts with others in the community; (2) frequency in entering community settings; (3) number of roles played. Although eventually there is some increased participation in these areas, the extent of this participation varies and is influenced by a number of factors. For example, physical limitations imposed by the injury and personality (pre-morbid and post-morbid; attitudes and behaviors of the nondisabled).

> All paraplegics face problems which evolve from the stigma of disability. In the hospital, medical personnel help paraplegics develop a self-image of independence and personal worth. Although difficulties are encountered, it is easier to establish and maintain this self-image in the sheltered social environment of the hospital than in the world outside. When paraplegics return to their homes and communities, definitions of their disability as a social stigma reach the height of salience. This common problem apparently orders their course of socialization (Cogswell, 1968).

In the United States, the body-whole and the body beautiful hold high social value. Therefore, both the disabled and nondisabled may regard the spinal cord injured individual as inferior (Littman, 1962). Handicapped people often see themselves as a

misunderstood minority, and the general population as lacking adequate information on many aspects of their disability (Goodman, 1971).

The evidence indicates the spinal cord injured, both paraplegics and quadriplegics, can become functioning, participating and contributing members of society. Many tasks and occupations do not necessitate the use of one's arms and legs, rather the use of the mind is required; and one's brain is not affected by a spinal cord injury (Maxwell, 1971).

At the present time, a spinal cord injured individual's quality of life is less than that of the normal population. The community must address itself to problems faced by the spinal cord injured in the areas of: independent housing, transportation, recreation, architectural barriers, employment and the participation of the spinal cord injured in a social life (Maxwell, 1971). For example, the Ruge (1966) study reveals that the majority of recreational activities pursued by a sample of the spinal cord injured population are individualized rather than social activities: i.e. reading and studying, arts and crafts, coin collecting, tropical fish, music.

Although physicians estimate only 3% of those currently living at Spinal Cord Centers, could be discharged to totally independent living in the community, there is no reason for these individuals not to participate in the community setting (VA, 1972). The need for an attendant does not necessitate the limitation of interactions and activities with the nondisabled population.

Functioning as a Member of the Family

A strong, unified, resourceful family is an extremely important contributor to the emotional, social and vocational recovery of a spinal cord injured individual (Travis, 1961). According to Slater, Sussman and Stroud (1970) "...for long-term recovery the family context has more to do with rehabilitation outcome than considerations of the patient's characteristics or conditions, continued therapy or spontaneous physical recovery."

Spinal cord injured individuals with aspirations to return to work or to the family appear to do best in rehabilitation recovery. Individuals, who prior to their injury were married or lived in households with parents, after onset of spinal cord injury participated more fully in the household division of labor than the unmarried patient and/or those living with kin other than parent or spouse. Furthermore, data indicate the greater a patient's premorbid participation in household activities, the greater the likelihood to achieve an independent level of ADL (activities of daily living) functioning earlier in the course of rehabilitation (Slater, Sussman and Stroud, 1970).

There is a greater willingness to accept a disabled individual back into the family home and work with him/her toward a favorable rehabilitation outcome, if he/she does not impose inordinate demands for time and emotional support from other members of the family. If the disabled person, for any reason, is a non-participator or makes excessive demands, there is a tendency to remove this member from

the home with a corresponding reorganization of roles and obligations within the new grouping (Slater, Sussman and Stroud, 1970).

It should be noted that a spinal cord injured individual cannot participate within the family circle quite as before. There are changes in the number and type of roles played (Cogswell, 1968). The male patient who might have been the "head" of the household prior to his injury, must at least for some period of time after his injury, (i.e., during physical recovery, during rehabilitation) accept a more dependent role.

Several studies indicate that role-reversal is a very serious problem for families coping with the effects of disability of one of its members. However, successful role reversals are possible if organization (managerial) aspects of homemaking are stressed in educational programs for the disabled and his/her family (Beppler, 1974).

Carpenter (1974) reports that disabled husbands assume more household tasks when their wives are employed, than when their wives are not employed. However, the degree of severity of the husband's disability affects the extent to which the husband assumes household duties. The mildly disabled (moves about by self without too much difficulty) participated in more household tasks than the severely disabled (moves about only with difficulty or needs the help of others).

VII. Technologies

I. Medical technologies:

1. testosterone to offset tissue breakdown.

2. urinary antiseptic to prevent urinary infection.

3. diet which is high in protein content; avoidance of foods which contribute to gastiointestinal discomfort.

4. a lifetime regime of ingesting enough water to reduce the likelifood of formation of kidney stones.

5. treatment to relieve bladder and bowel distension; an indwelling catheter is introduced to keep the bladder empty or intermittent catheterization can be another successful method. Both forms of treatment require the prevention of an inflammatory response to the passage of the tube. The bowel is similarly emptied regularly. After the acute medical problems have been controlled, the patient can learn a number of bowel and bladder control techniques depending upon the extent of his/her injury.

6. treatment of decubitis ulcers; patients should be turned every two hours and the skin in the pressure areas massaged lightly. After the acute period, the patient may be trained to shift his/her support from time to time while sitting to prevent ulceration.

7. antipyretic measures may be employed to control the fever outbreaks which result from impairments in body temperature control.

8. the immediate goal for hospitalized patients should be to get them in a standing position as soon as possible. This can be accomplished by the use of a tilt board. These are a number of benefits derived from a shift from the bed position to the standing position--the hazards of urinary infection and renal calculi are lessened; circulation is improved and protein utilization is restored. These improvements are mutually reinforcing, i.e. improved circulation aids in counteracting ulceration.

II. Techniques for lessening severe spasticity:

1. drugs

2. stretching

3. electrical stimulation

4. braces

5. tranquilizers

6. peripheral operations (neurotomies, myotomies, tenotomies)

7. local injections

8. rhizotomies

III. Orthotics: "application of exoskeletal devices to limit or assist motion of any given segment of the body as an analogous mechanical substitute" (Goldfinger and Goodgold, 1975).

1. plastic opponens orthosis (modular unit now available in kit form)

2. reciprocal wrist extension Finger Flexion orthosis.

3. orthotic research arm (computer-controlled device not at clinical level of application, Reswick, 1970).

4. Ranchos Los Amigos electric arm (requires tongue switches) (Allen, 1970)

5. arm othoses utilizing CO_2 powered devices (Engen, 1969).

6. electrically driven (Battery-powered) hand orthoses (Lehneis, 1968)

7. ankle foot orthoses (AFOs) (CPRD, 1972c)

   a. posterior leaf spring AFO

   b. spiral AFO

   c. hemispiral AFO

   d. posterior solid ankle AFO

   e. anterior leaf AFO (University of Alabama, Birmingham RT-19)

8. long-leg brace (with double bar uprights, knee locks, and a 90 degree position stop ankle joint).

9. plastic laminated thigh-knee-ankle orthoses (Lehneir, 1972). This device provides maximum function with a minimal expenditure of energy for the patient.

10. TIRR polypropylene orthoses (RT-4 Baylor College of Medicine and the Texas Institute for Rehabilitation and Research)

11. IRM balanced for arm orthosis (BFO)

12. short leg (ankle) braces: for use below L-4 injury

13. crutches: for use below L-4 injury

14. "feeders" for elbow and shoulder support, especially in the early stages of rehabilitation.

15. electric motor splints.

16. leather cuffs strapped to hands (drives fingers into flexion)

17. Rancho Los Amigos flexor hinge splint

18. springs to open hand with electric motor to provide "voluntary" closure

19. balanced forearm orthoses: mobile arm supports; feeders.

20. multi-axis device which drives the entire arm through geared electric motors.

IV. Adaptive self-help devices: "additions or extensions to function, adapted to patients for performance of specific tasks and activities." (Goldfinger and Goodgold, 1975).

1. activities for daily living aids for personal grooming and self-care

2. hand controls for automoblies

3. adaptation of the controls on small, inexpensive tape recorders

4. adaptation of standard telephone equipment (Sullivan, 1968)

5. voice operated telephone (Sullivan, 1968)

6. IRM ballbearing arm support.

7. communication system for use by quadriplegics: morse code will be translated into ASC II code (a standard electrical code for communication) for controlling a typewriter, tape recorder, microfilm reader and telephone.

8. electric wheelchairs

    a. proportionally controlled electric wheelchairs

    b. reclinable wheelchairs (C4 and C5 level injury patients) to permit non-weight bearing rest periods during the day

    c. removable armrests on wheelchairs (Long, 1971)

    d. detachable, electrically powered unit for a wheelchair (Ewing, 1969)

    e. eye-movement controlled wheelchair (Baylor College of Medicine and the Texas Institute for Rehabilitation and Research)

9. special seats designed to prevent decubitus ulcers

10. clamp devices for emptying urinals bags

11. special control systems: tongue switches; touchplates; microswitches

12. mouthstick: useful in typing, writing, dialing and turning pages.

13. overhead sling suspension: interim measure if permanent feeders are unnecessary

14. hydralic lift: to move the patient from bed to wheelchair

15. beds: adjusted to wheelchair height

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CHAPTER 3
DISABILITY ANALYSIS: CEREBRAL PALSY

I. Definition

> "Cerebral palsy embraces the clinical picture created by injury to the brain, in which one of the components is motor disturbance. Thus, cerebral palsy, may be described as a group of conditions, usually originating in childhood, characterized by paralysis, weakness, incoordination or any other aberration or motor function caused by pathology of the motor control centers of the brain. In addition to such motor dysfunction, cerebral palsy may include learning difficulties, psychological problems, sensory defects, convulsive and behavioral disorders of genetic origin" (United Cerebral Palsy Research & Education Foundation, 1958).

"Cerebral" means "brain" and "palsy" means a "motor disability," thus, cerebral palsy refers to a motor disability caused by brain dysfunction (Kirk, 1972). Cerebral palsy was first described medically in 1861 by the English surgeon, William J. Little as a spastic syndrome in children. Formerly associated only with motor disability, it has now come to be considered as one of the most complicated disabilities known as it can affect so many capacities. Cerebral palsy is a multiple crippler (Kirk, 1972; Keats, 1965).

Cerebral palsy is basically a physical disability resulting from intracranial lesions. Though the general physical symptoms of motor disability may be far more obvious clinically, associated disabilities may be present to a greater or lesser extent, depending on the location of the lesion or lesions and degree of involvement. Manifestations of the disorder differ according to the setting and extensiveness of the lesion and may vary from mild motor incoordination to virtually complete helplessness. In some cerebral palsied children, only one disability factor may appear while other may mainfest any combination of factors (Cruickshank & Rauss, 1955; Keats, 1965; Wilson, 1974; Perlstein, 1952; Kirk, 1972).

Given the complexity of the brain, any injury or lack of development in it may give rise to a variety of disturbances. Cruickshank & Rauss (1955) emphasize that cerebral palsy is only one component of a broader brain damage syndrome. Though the basic characteristics of the cerebral palsied child are paralysis, weakness, incoordination, there may exist other symptoms which reflect a damaged brain. These include convulsions, mental retardation or deficiency, visual, hearing and other perceptual problems, as well as speech, behavioral and emotional disorders (Cruickshank & Rauss, 1955; Perlstein, 1952). The underlying cause of these symptoms is brain damage but not of a specific type. Similar pathological findings are found in such clinical entities as mental retardation, epilepsy, and other behavioral disorders of organic origin (Cruickshank & Rauss, 1955).

Keats has defined cerebral palsy as a "non-progressive complex clinical entity with a multitude of handicaps" (Keats, 1965). The non-progressive nature of cerebral palsy should be emphasized. Under the present definition, a child with a progressive brain lesion is not diagnosed as having cerebral palsy until the progressive nature of the etiology has been eliminated. Progressive diseases and those characterized by transient motor disturbances are excluded from the definition, as well as those primarily the result of spinal cord lesions (Keats, 1965; Balk & Ingram, 1955). Only that damage to the central nervous system that affects motor coordination and occurs during the development years is classified as cerebral palsy. Therefore, damage later in life that impairs already established normal motor function is not considered a cause (Wilson, 1973).

Cerebral palsy may be considered a disease, but not in the ordinary sense. Swartz (1951) describes cerebral palsy as a long-term, nonfatal, noncurable

disease which is amenable to therapy and training. It is nonfatal per se. It is not contagious or epidemic in form, and is considered a disease primarily as a neurophysical and neuropsychological deviation (Cruickshank & Rauss, 1955). It might best be described as a well-defined condition characterized by a group of concurrent neuromuscular disabilities which are characterized by distrubances of voluntary motor function, especially of the extremities (Wilson, 1973). By nature of its describable syndrome, it is consistent in some ways to other diseases (Kirk, 1972).

Classification of Types of Cerebral Palsy. A classification of the types of cerebral palsy satisfying all demands and criteria is difficult (Keats, 1965). Classification is generally based upon the particular kind of motor involvement, but there are also systems that use other criteria such as degree of location of the physiological impairment (Crothers & Paine, 1958; Wilson, 1973; Minear, 1956; Perlstein & Barnett, 1952). Ordinary medical criteria are almost impossible to apply because there exists no single causal etiology; no characteristic course the condition takes; and because the accurate description of motor patterns may not give any evidence of other important elements such as the related intellectual and emotional disorders (Keats, 1965).

Cerebral palsy is also classified with regard to the region or regions of the body in which muscular dysfunction occurs. From this classification system are derived the terms paraplegia (associated with involvement of the legs only), quadriplegia (involving all four extremities), hemiplegia (involving the arm and leg of one side of the body), and monoplegia (involving only one extremity) (Allen & Jefferson, 1962). Relatively few cerebral palsy victims are monoplegic. Hemiplegia is found to the greatest extent among

spastics. Both athethoids and spastics manifest quadriplegia, with spastics usually showing greater involvement of the legs, and athetoids, of the arms (Kirk, 1972). In describing cerebral palsy, as in describing other diseases, the diagnostic team normally takes into consideration the general condition, the parts of the body affected, the degree of involvement, such as mild or severe, and associated disabilities. Thus, a cerebral palsied child may be described as a moderate, right hemiplegic, with accompanying associated disabilities (Kirk, 1972).

The most common type of classification is based on the predominant clinical manifestations determined by neurological signs and their topographical distribution. Based on this criteria the most widely accepted categories of cerebral palsy have been designated as follows: spasticity, athetosis, ataxia, rigidity; tremors, and mixed conditions (Kirk, 1973; Abbot, 1957; Wilson, 1973; Bakwin & Bakwin, 1972; United Cerebral Palsy Association, 1958; Education & Research Foundation, 1958).

These types will be briefly defined below and discussed in more detail later in this discussion according to particular etiologies associated with the type, incidence and prevalence of each type among the estimated total cerebral palsy population, and associated disabilities according to type and life function area they affect.

Spasticity. The largest number of cerebral palsy victims manifest this condition. Descriptively, the spastic is usually identified by "stiffness" of the musculature (Allen & Jefferson, 1962). The condition is further characterized by contracted muscles, hyperactive reflexes, hypertonicity, clonus and exaggeration of the stretch reflex in the affected parts (Bakwin & Bakwin 1972; UCPA, 1958; Allen Jefferson, 1962; Kirk, 1972). It may occur

in one or more limbs with hemiplegia being the most common locus of involvement (Kirk, 1972). The gait is knock-kneed and pigeon-toed (Abbot, 1975).

Spasticity is caused by lesions in the cerebral cortex, the pyramidal tracts, and possibly in the extrapyramidal tracts. The areas of the brain which deal with involuntary movements are defective. Normally these areas suppress certain impulses, contractions, and stimulations. When one or more areas are affected, suppression does not occur, resulting in a state of tension or spasticity of the muscles, an absence of balance, and jerky, uncontrolled movements (Kirk, 1972).

Athetosis. The second largest group of cerebral palsied children suffer from this condition. It is caused by a lesion on the basal ganglia and extrapyramidal tracts of the brain (Wilson, 1973). The condition involves only the joints of the body and results in an inability of gross directional control, uncontrolled, jerky, irregular twisting, or wormlike movements, marked incoordination and almost constant, purposeless motion (Bakwin & Bakwin, 1972; Kirk, 1972; Abbot, 1957). The gait is scissors-like (Abbot, 1957).

Ataxia. This condition is less prevalent than the two precedint types, but estimates of its occurrence vary considerably (Bakwin & Bakwin, 1972; Wilson, 1973). It is primarily a disturbance of automatic balance (Abbot, 1957). It is caused by a lesion in the subcortical area, most likely in the cerebellum which normally controls balance and muscle coordination. Movement is uncoordinated and reeling, much like that of a drunken person (Bakwin & Bakwin, 1972). Sometimes nystagmus (jerky eye movement) is common (Kirk, 1972). Head turning is common but should not be taken to imply inattentiveness, as it is a typical clinical picture (Abbot, 1957).

Ordinarily, ataxia is not detected at birth, but is apparent when grasping and walking begin. There are varying degrees of ataxia, as there are in the other types, ranging from mild (barely detectable) to severe. Severity of impairment depends on the extent of damage to the cerebellum (Kirk, 1972; Bakwin & Bakwin, 1972; Andrews, 1972; Wilson, 1973).

Rigidity. This is a relatively rare form of cerebral palsy. The child with this condition manifests an interference with postural tone, disturbances of the agonist-antagonist relations with resistance to passive motion of both agonist and antagonist muscles (Keats, 1965). If the resistance to passive motion is continuous, it is called "lead-pipe" rigidity; if discontinous, it is referred to as "cog-wheel" rigidity (Keats, 1965).

It is thought to be caused by a very diffuse brain lesion involving a wide area (Abbot, 1957). The type resembles the severe spastic but there is no stretch reflex. The ataxic has difficulties in moving through a complete range of motions (Abbot, 1957).

Tremor. This, too, is a rare form of cerebral palsy. Stemming from damage to the basal ganglia, the condition's salient characteristics are rhythmic involuntary movement of an irregular nature which results from interference in the normal balance between antagonistic muscle groups (Keats, 1965; Wilson, 1973; Bakwin & Bakwin, 1972; Kirk, 1972). The tremor type is further subdivided into two categories. The intention tremor involves contractions occuring only when motion is attempted. The non-intention tremor type manifests contractions at all times (Abbot, 1957).

Mixed types. Mixed type do occur, though, infrequently. Accurate differential diagnosis and classification are difficult. Categories are spastic athetosis, rigid spastic, and spastic ataxic (Keats, 1965).

II. Societal Characteristics of the Cerebral Palsied

The literature provides prevalence, incidence, mortality, and cost information.

Prevalence. Estimates vary according to definition and classification, the area of study, the age group studied and other factors (Wilson, 1973). Mild cases may go undiagnosed or undetected, or if the condition is associated with other disabilities, such as mental deficiency, the children tend to be enrolled in institutions specifically for that disability and are so classified (Kirk, 1972). Robinault and Denoff (1973) estimate that one out of seven persons with cerebral palsy never come to clinics because disabilities are sometimes so mild that they can fit into normal society.

Though figures vary according to course, estimates of the total cerebral palsy population are between 550,000 (Bakwin & Bakwin, 1972) to 750,000 (Robinault & Denoff, 1973; United Cerebral Palsy Association, 1958); while Molnar and Taft (1973) and Research and Education Association (1973) estimate 700,000. The United Cerebral Palsy Association who estimate prevalence at 750,000 believes that of the total cerebral palsy population, about one-third are teenagers and young adults.

Wilshik (1956) in a study of handicapped children in Georgia estimated that five out of every 1,000 persons under 21 years have cerebral palsy. Friedman and MacQueen (1971) estimated 1.7 per 1,000 persons are cerebral palsied individuals in need of special education. The discrepancy between these two figures might be attributed to the fact that not all cerebral palsy children require special education.

Other studies estimate prevalence between 1.6 and five per 1,000 population under 21 years of age (Molnar & Taft, 1973; Bakwin & Bakwin, 1972; Wilshik, 1956; Phelps, 1950; Altman, 1955; Wolfe & Reid, 1958). The average

of the various estimates is around three per 1,000 population which is probably a more accurate reflection of the present situation (Wilson, 1973).

Incidence. Woods (1963) noted an apparent overall lowered incidence trend and an increase in milder, subclinical cases than has been noted previously. The factors responsible for such trends are yet to be determined. Kershaw (1969) suggests that the increase in milder cases of cerebral palsy is possibly due to parental awareness of the condition and increased hope that something can be done.

The United Cerebral Palsy Association (1958) estimates incidence at 15,000 babies born each year with cerebral palsy--one in every 200 live births. Other estimates range between 3.24 per 1,000 population (Wolfe & Reid, 1958) and 5.9 per 1,000 population (Wishik, 1954), with an average around 4.5 per 1,000 population. Studies of incidence according to types of cerebral palsy vary considerably. A summary of several studies appears in Table 3-1.

Mortablity. Cohen and Mustacchi (1966), in a study of 1,416 cerebral palsied children over a 10 year period concluded that cerebral palsied patients under 21 years of age have eight to nine times the mortality rate of the general population in that age group. Perlstein (1953) has estimated the life expectancy of the cerebral palsied person at 32 years. Highest mortality is displayed in the group with multifocal damage which results in a mixed type of cerebral palsy (Cohen & Mustacchi, 1966). However, the survival rates in athetoid, ataxic, unspecified, and mixed varieties of cerebral palsy are not significantly different from one another. Each one, however, and all cumulatively are significantly different from the values observed for the group with spastic cerebral palsy. The greatest survival rate is displayed among individuals with the spastic type of cerebral palsy (Cohen & Mustacchi, 1966). Bakwin and Bakwin (1973) estimates that one-third of

TABLE 3-1

INCIDENCE BY TYPES OF CEREBRAL PALSY

Source	Spasticity	Athetosis	Ridigity	Ataxia	Tremor	Mixed
Bakwin & Bakwin, 1972	50%	25%	15%	9%	1%	1%
Molnar & Taff, 1973	50-65%	20-25%	5-10%	1-10%	Rare	--
Hopkins, et. al., 1954	45.9%	23.7%	12.6%	10.8%	--	--

children born with cerebral palsy are so severely affected that there exists little potential for improvement. Life expectancy is less than ten years.

Costs. The United States Department of Health, Education and Welfare estimates the annual cost of care for cerebral palsy at $1.6 billion (See sheet called Cerebral Palsy - Facts & Figures, U.C.P. Association, Inc., 1975).

III. Demographics

There are no apparent economic, social or geographic predilections of cerebral palsy (Bakwin & Bakwin, 1972; McCarroll, 1972). Cerebral palsy, however is associated with childhood, as it is a developmentally disabling condition. About one-third of cerebral palsy sufferers are under 21 years of age, and the association of cerebral palsy with prematurity is well known (Bakwin & Bakwin, 1972). At least 40 percent of all persons with cerebral palsy have been born prematurely. Further, data indicate cerebral palsy is more frequent in children born to older women (McCarroll, 1972).

The occurrence of cerebral palsy is higher among white children than blacks (McCarroll, 1972).

IV. Etiology

There is no single causal etiology of cerebral palsy. Multiple possible factors are involved in most retrospective histories, and it is often difficult to distinguish cause from effect since there is no adequate way of supplying controls which would permit firm conclusions. Adequate statistical evaluation of the etiology of cerebral palsy would require a very large number of highly detailed obstetrical histories as well as very carefui follow-up studies (Keats, 1965). For clarity, etiological factors are usually divided arbitrarily into these major groups: prenatal, natal, and postnatal. It

is also possible that the proportion of etiologic factors is somewhat different in the different types of cerebral palsy (Keats, 1965). Table 3-2 shows common clinical correlates in cerebral palsy.

Prenatal factors. These factors operate from the time of conception to the onset of labor. Perlstein (1952) states that prenatal factors are responsible for approximately 30 percent of all cases of cerebral palsy. Prental factors include hereditary or genetic defects and injuries acquired by the developing fetus in the uterus (Perlstein, 1952; Keats, 1965).

Hereditary causes are conditions that are genetically transmitted and may involve racial or familial predilection and are often sex-linked. Russ and Soboloff (1958) have included in this category such familial diseases as familial athetosis, congential tremors, familial spastic paraplegia, atonic diplegias, and familial rigidities. In these conditions the basic pathology is genetically transmitted developmental defects in the basal nuclei (Keats, 1965).

Other progressive symptoms that may or may not be manifest at birth but may appear later in life are sometimes included as causes, but there is some disagreement on this point. Perlstein (1952) includes all Tay-Sacks disease, tuberous sclerosis, demyelinating diseases, and other rare neurological conditions as being among the causes of cerebral palsy. Keats (1965) arques these conditions should not be included as etiological factors due to their progressive nature. As has been noted previously, cerebral palsy is usually defined as involving non-progressive lesions in the brain.

Other studies have found that children with cerebral palsy present a higher incidence of other congential malformations such as extra digits and cleft palate. It is suggested that the high incidence of prematurity in

TABLE 3-2

COMMON CLINICAL CORRELATES IN CEREBRAL PALSY

History (Precipitating Factor)	Common Neurological Sequela
Prematurity	Spastic Paraplegia
Breech Delivery	Athetoid or Spastic Paraplegia
Toxemia of Pregnancy	Spastic Hemiplegia
Birth Trauma	Spastic Quadriplegia or Hemiplegia
Anoxia	Athetoid or Rigidity
Rh Factor	Athetoid with Deafness and Paralysis of Supravision
Maternal Rubella	Spastic with Auditory Aphasia or Deafness, Cataract, Congenital Heart
Precipitate of Caesarian Delivery	Spastic Quadriplegia

Source: Perlstein, M. A. Infatile Cerebral Palsy: Classification and Clinical Correlations, JAMA, 1952, 149(1).

cerebral palsy would partially explain this (Eastman & DeLeon, 1955; Keats, 1965).

Prenatal etiologic factors acquired in the uterus are varied. They include blood-type incompatibility, especially with the Rh factor and other forms of iso-immunization (Perlstein, 1952). Due to improvements in health education and transfusion techniques, this factor is now responsible for less than three percent of all cerebral palsy cases (Perlstein, 1952). Maternal infections, particularly rubella and occasionally other virus diseases may cause cerebral palsy. Other prenatal causes include prenatal anoxia; cerebral hemorrhage, usually caused by maternal toxemis of pregnancy; metabolic disturbances, including all maternal diabetes; and gonadal irradiation, which is a mutation in the developing ovum due X-ray therapy (Perlstein, 1952; Wilson, 1973).

Kernicterus ("nuclear jaundice"). This is also another important prenatal cause of cerebral palsy. It may produce athetosis, deafness, mental retardation, and auditory aphasia. The large amount of research devoted to this condition during the past 25 years has resulted in its more successful treatment both during pregnancy and during the immediate post-natal period. This syndrome was once thought responsible for 8-10 percent of all athetoids, but this figure has now been reduced to as low as 1-2 percent (Keats, 1965).

Natal or paranatal factors. These factors operate from the time of onset of labor to the time of viability of the delivered child. These are the most frequent causes of cerebral palsy and account for approximately 60 percent of all cases (Perlstein, 1952). Most authors agree that the most common cause of cerebral palsy is prematurity. This is because a child

who is prematurely born is more liable to brain injury (Keats, 1965). Illingworth (1958) noted that 33 percent of all cerebral palsy individuals were prematurely born.

Natal factors. These factors include anoxia, which is one of the most important causes of cerebral palsy. It may be caused by a variety of factors including atelectasis (incomplete expansion of the lungs), mucus obstruction, or breech deliveries. Cerebral hemorrhage and contusions, the commonest cause of injury to the brain during delivery, may be caused by prolonged labor, difficult or abnormal birth, precipitant birth, excessive birth weight, sudden pressure changes, as during caesarean delivery, vitamin K deficiency or anemia; and trauma to the head during delivery, such as in forceps delivery. A relatively small number of brain injuries can be attributed to the latter cause (Wilson, 1973; Perlstein, 1952).

Postnatal factors. These factors operate during the early years of life account for less than 10 percent of all cases of infantile cerebral palsy (Perlstein, 1952). The factors include mechanical damage to the brain (traumatic) infections of the brain tissue, such as encephalitis and meningitis, toxic causes, such as lead poisoning, or poisoning due to arsenic, or coal tar derivatives. Cerebral anoxia, or insufficient oxygenation of the brain due to carbon monoxide poisoning, and high altitude anoxia are also causes of cerebral palsy in the postnatal period (Wilson, 1972; Perlstein, 1952; Keats, 1965).

V. Life Functions

The cerebral dysfunction resulting in cerebral palsy may also cause a variety of other handicaps, singly or in combination (Kirk, 1972). Though, cerebral palsy is a condition whose major clinical syndrome is motor disability,

all of the life function areas may be affected, especially for those individuals suffering from moderate to severe cases of cerebral palsy. The degree of impairment and the presence of associated disabilities is dependent upon the location and extensiveness of the brain lesion or lesions.

It has been estimated that from 50-75 percent of all cerebral palsied patients display some kind of speech impairment; mental retardation is suspected in about 50 percent; approximately the same percentage are believed to have visual problems; hearing disorders and convulsive disorders are present in from 25-35 percent, and behaviorally, more than 70 percent conduct themselves as if they were intellectually retarded (Allen & Jefferson, 1962; Andrews, 1972; O'Reilly, 1971; Scherzer, 1974). Also associated with cerebral palsy are problems of social and emotional adjustment of the child and his family (Allen & Jefferson, 1962).

Mobility. Table 3-3 describes the symptoms which restrict the cerebral palsy patient in the area of mobility. The leading clinical syndrome of cerebral palsy is a motor deficit (Molnar & Taft, 1973). In the course of neurologic maturation of normal infants and young children there is an orderly sequence in the dissolution of primitive postural reflexes. It has been postulated that the motor dysfunction in cerebral palsy is characterized by a persistence and preponderance of these primitive reflexes, predominantly tonic neck and tonic labryinthine patterns. They, in turn, interfere with the appearance and effective action of those automatic reactions, primarily the righting equilibrium reactions that are necessary for purposeful motor activities (Molnar & Taft, 1973).

Difficulties in the early recognition of cerebral palsy are due to the fact that cortical control of movements does not occur until later in infancy,

TABLE 3-3

LIFE FUNCTION TABLE - CEREBRAL PALSY: MOBILITY

Statement of Problem	Dynamics	Source
Little movement in lower extremities.	Ataxic, infancy	Andrews, 1972.
Marked resistance to passive motion.	Rigidity	Bakwin & Bakwin, 1972.
Difficulty controlling sitting, standing, and walking without the support of crutches, braces, etc. Postural and tone abnormalities.	Spasticity (severe cases) All types, but especially ataxic	Kirk, 1972. Abbot, 1957.
Frequent falls caused by poor coordination.	Ataxic	United Cerebral Palsy Research & Education Foundation, 1958.
Lack of synchronization of muscle groups inhibiting smooth movements.	All types	Wilson, 1973.
Knock-kneed, pigeon-toed gait.	Spasticity	Abbot, 1957.
Inability to close lips and therefore retain food.	Infancy and young child, athetosis	Scherzer, 1974.
Contractions at all times.	Tremor (Non-Intentional)	Abbot, 1957.
Contractions when motion is attempted.	Tremor (Intentional)	Abbot, 1957.
Difficulties in moving through a full range of motion.	Rigidity	Abbot, 1957.
Motor output inaccurate due to disturbance of sensory intake.	Ataxia	Abbot, 1957.
Involuntary eversion and inversion of legs.	Athetoid	Keats, 1965.
Difficulties in bending joints.	Athetosis, spasticity	Abbott, 1957; Allen & Jefferson, 1962.
Tenseness in stretch reflex (called the "Stretch reflex").	Spasticity	Morgenstern, 1964.

Table 3-3 cont'd.

Statement of Problem	Dynamics	Source
Difficult, inaccurate voluntary motion.	Spasticity	Morgenstern, 1964.
Incoordinate action of the extrinsic ocular muscles causing strabismus	Spasticity	Abbott, 1957.
Inability of gross directional control.	Athetosis	Abbott, 1957.
Almost constant motion of extremities.	Athetosis	Morgenstern in Hellmuth, 1964.
Unorganized muscular movements.	Athetosis	Abbott, 1957.
Excessive involuntary motion which involves facial and speech muscles.	Athetosis	Abbott, 1957.
Spasms of laughter or crying with no emotional correlates.	Tremor	Abbott, 1957.
Widespread continuous muscle tension or "lead-pipe" stiffness.	Rigidity	Keats, 1965; Morgenstern, 1964.
Extensor thrust in feet.	Rigidity	Abbott, 1957.
Crossed legs of scissors position.	Rigidity	Abbott, 1957.
Inability to sense body position in gravitation field.	Ataxia	Abbott, 1957.
Primarily a balance loss.	Ataxia	Abbott, 1957; Morgenstern, 1964; Bakwin & Bakwin, 1972.
Involvement of nystagmus (vibration) oscillatory movement of the eyeballs.	Ataxia	Abbott, 1957.
Head drawn back, extended tense neck, with mouth held open.	Athetosis	Kirk, 1972.
Lurching, stumbling gait.	Athetosis	Kirk, 1972.
Uncontrolled postural attitude likened to writhing and wriggling.	Athetosis	Kirk, 1972.
Constant shaking making use of hands and feet difficult.	Tremor	Morgenstern, 1964; Bakwin & Bakwin, 1972; Kirk, 1972; UCPA, 1958.

there are however, some early signs and symptoms. These include slow motor development, unusual patterns of motor development, persistence of primitive reflexes, and abnormal muscular tone. Normal infants are ambidextrous until about 18 months, and a younger baby who seems to prefer one hand should be suspected of having a motor deficit of the opposite extremity. General observation of a baby's movements and muscle responses is the most reliable technique for detecting cerebral palsy (Andrews, 1972).

Other early symptoms of motor deficit include poor sucking with tongue thrust, a tendency to opisthotonos posture exaggerated with crying, delay in attainment of motor milestones especially if social, personal, and language behavior are normal. Floppiness of hypotonia and stiffness and resistance encountered when placing the infant in a sitting position may be early indicators of spasticity. Difficulty in diapering the infant due to scissors-like motion of the lower extremities may also indicate spasticity. Atypical or asymmetrical crawling and a persistent tendency to stand on the toes are other symptoms of a possible motor deficit (Molnar & Taft, 1973).

Problems are often encountered in caring for the infant with motor dysfunction. Dressing can be a particular problem if the mother attempts to clothe a hypertonic child who has a strong tonic neck pattern. There are problems in bathing when a child needs complete support for sitting and is obviously in an insecure position. Postural and tone abnormalities are associated with many difficulties in sitting. Feeding presents the most complex variety of problem in management and learning in the affected child. The child is, by necessity, a passive recipient in the feeding process, possibly deriving limited nutrition and little stimulation from the experience. The child may be unable to close his lips and retain food because

of inadequate oral development and persistence of infantile reflexes. Although the child may be well-supported in a high chair, he may be in an extension posture. Moreover, continuous tongue thrusting and an asymmetric tonic neck pattern may present problems in body and trunk control affecting the child's ability to perceive and benefit from the stimulation of the feeding process (Scherzer, 1974).

The various motor manifestations differ according to type of cerebral palsy. Spasticity is characterized by the involuntary contraction of the affected muscles when suddenly stretched (stretch reflex) which results in tenseness, and difficult, inaccurate voluntary motion (Morgenstern, 1964). Motions of the extremities are made slowly and with great effort. When attempting to bend the various joints, opposing muscles contract, blocking the patient's efforts (Allen & Jefferson, 1962).

In mild cases, a child may extend his arms for balance. In moderate cases, a child may hold the arms close to the body in an effort to control the scissoring-type walking movement. In the more severe cases, a child may have such poor control over his body that sitting, standing, and walking become difficult or impossible without the support of crutches, braces, or a walking frame. Spasticity may occur in one or more limbs, but hemiplegia is the most common locus of involvement (Kirk, 1972).

Athetosis is characterized by the involuntary contraction of successive muscles resulting in marked incoordination and almost constant motion of extremities (Morgenstern, 1964; Abbot, 1957; Keats, 1965). The movements are often slow, wormlike, and purposeless (Bakwin & Bakwin, 1972).

The head is frequently drawn back, with the neck extended and tense. The mouth may be held open, causing drooling and a protruding tongue. The

gait is lurching, writhing, and stumbling in manner. Movements are not rhythmical and the postural attitude is uncontrolled with a tendency to writhe and wriggle (Kirk, 1972).

The athetoid can put his hand to his mouth, but in doing so he may sometimes go through various uncontrolled movements (Andrews, 1972). In extreme cases, he may manifest squirming gestures and marked facial grimaces. The writhing and squirming motions, however, do not occur during sleep, but only during the conscious state. As conscious effort and emotionality increase, the movements become intensified (Kirk, 1972). Phelps (1958) differentiated several types of athetosis based on the particular muscles and motions involved. In the rotary type, the head may turn from side to side or the legs may evert and invert involuntarily. Other types categorized by Phelps include the tension, non-tension, dystonia, and tremor athetosis.

The principal motor manifestation of ataxia is a disturbance of balance and equilibrium resulting in uncoordinated movement, impaired balance, and lack or proper orientation in space (Bakwin & Bakwin, 1972). The gait is like that of a drunken person with the patient walking with a high step (Bakwin & Bakwin, 1972; Kirk, 1972). Poor coordination causes the ataxic to fall easily (United Cerebral Palsy Research and Educational Foundation, 1958).

In the ataxic, the reflexes are hypoactive and horizontal nystagmus (jerky eye movement) in usually present. The ataxic infant shows little movement in the lower extremities (Kirk, 1972; Andrews, 1972).

In the rigid type of cerebral palsy, the muscles are hypertonic and there is marked resistance to passive motion (Bakwin & Bakwin, 1972). This type is similiar to severe spasticity, but there is no stretch reflex. The

child also experiences a heavy "lead-pipe" stiffness not encountered in spasticity (Keats, 1965; Abbot, 1957?. An individual with this type of cerebral palsy will find it difficult to move through a complete range of motion (Abbot, 1957). Motion is more diminished than abnormal (Kirk, 1972).

Tremor cerebral palsy results from an interference in the normal balance between antagonistic muscle groups and is characterized by rhythmic, involuntary, incontrollable motions limited to certain msucle groups (Bakwin & Bakwin, 1972; Kirk, 1972; Morgenstern, 1964). When contractions occur only when motion is attempted, it is termed intention-tremor; if contractions occur at all times, the condition is called non-intention tremor (Abbot, 1957). Constant shaking makes use of the hands and feet difficult (United Cerebral Palsy Research and Education Foundation, 1958).

Health. Table 3-4 summarizes some of the complications of the condition which affect the health of the cerebral palsy victim. It has been estimated that from 25-35 percent of the cerebral palsy population suffer from seizures, though seizures have been found to occur with the greatest frequency among spastics (Gordon, 1975). Children with the rigid types of cerebral palsy have a high incidence of convulsions. The incidence of seizures among hemiplegics is around 67%; among paraplegics, 31%; and among quadriplegics 56% (Gordon, 1975) (see Table 3-5).

Difficulties in taking care of oneself and in personal hygiene imposed by the extreme motor deficits can be appreciated. The problems of bathing, dressing, and feeding that are present from infancy may continue into adulthood depending on the particular motor involvements, their amenability to therapy and training, type of treatment, and age at which treatment is begun.

TABLE 3-4

LIFE FUNCTION TABLE - CEREBRAL PALSY: HEALTH

Statement of Problem	Dynamics	Source
Seizures in 25-35% of total cerebral palsy population.	Occur with greatest frequency among spastics	Gordon, 1975.
Difficulties in personal hygiene (bathing, dressing, feeding) due to motor disabilities.	Moderate-Severe, all types; present from infancy through adulthood	Scherzer, 1974.
Excessive weight gain due to Point-Counterpoint phenomena.		McCarrol, 1972.
Nausea due to severe loss of sense of balance.	Ataxic	Abbot, 1957.
Frequent falls, poor coordination and shaking of hands and feet increases susceptibility to accidents.	Ataxic	United Cerebral Palsy Association, 1958.
Drowsiness caused by anti-convulsant drugs		Denoff, 1966.
Loss of appetite, complete lethargy, cramps, due to reactions to "Behavioral" Drugs.		Denoff, 1966.
Lassitude, nasal congestion, due to reaction to Relaxant Drugs		Denoff, 1966.
Dental problems caused by abnormal musculature.		Denoff, 1966.

Table 3-5

FREQUENCY OF SEIZURES IN VARIOUS TYPES OF CEREBRAL PALSY

Cerebral Palsy Type	Percentage of Each Type in Total Group	Percentage with Seizures
	N=1406	N=1265
Spastic	45.9	28.2
Athetoid	23.7	20.8
Rigidity	12.6	41.9
Ataxic	10.8	36.3
ALL GROUPS		29.2

Source: Hopkins, T. W., Bice, H. W. and Colton, K. C. Evaluation and education of the cerebral palsied child. Washington, D.C.: Council for Exceptional Children, 1954.

In cerebral palsy patients who manifest the Point-Counterpoint phenomenon, excessive weight gain is common due to the extreme limitation of activity caused by the condition. The child may try to move his arm in one direction, but the counter stretch of another muscle blocks its action, thus, severely limiting activity (McCarroll, 1972).

Ataxic patients may complain of nausea due to the severe loss of sense of balance (Abbot, 1957).

Various relaxant and "behavioral" drugs are used in the treatment of cerebral palsy. Side effects vary according to the particular drug used and individual constitution. Though most drugs used in the treatment of cerebral palsy are non-toxic and non-habit forming, adverse side effects do occur (Denoff, 1966).

Dexedrine and Benzedrine have been used with some success to counteract poor attention span, hyperactivity, mood swings, and variable behavior. Adverse effects of these drugs include loss of appetite, complete lethargy or withdrawal, excessive crying, or abdominal cramps (Denoff, 1966).

Dexedrine and Benzedrine are also sometimes used to offset drowsiness caused by anticonvulsants (Denoff, 1966).

Benadryl, used to alleviate hyperkinetic behavior may produce drowsiness. Thorazine and Serpasil, used to reduce anxiety, sometimes cause nasal congestion and lassitude (Denoff, 1966).

Abnormal musculature also may cause problems with the teeth. Major diseases and deformities are dental cavities, inflamation, and hypertrophy of the gingiva, destruction due to grinding deformity of the arches, and malocclusions. Despite these disorders, getting proper dental treatment for the cerebral palsy patient poses a greater problem than the dental disability itself. Sometimes the child is hospitalized and all necessary treatments are performed at one time under general anesthesia (Denoff, 1966).

Communication. Problems of vision, hearing, speech, and visual-motor perception are often present in cerebral palsy patients. (See Tables 3-6 and 3-7). All studies of visual deficiencies among cerebral palsy individuals have shown a large number suffer from visual anomalies (Kirk, 1972). Scherzer (1974) estimates that 50 percent of the cerebral population have vision problems usually due to a variety of eye muscle abnormalities. In a 1954 study of 1,297 cases, Hopkins, Bice, and Colton found 72.4 percent to have normal vision; the remaining 27.6 percent had defective or questionable vision. Of the visually defective sample, 47.7 percent were ataxic, 27.3 percent were spastic; and 20.4 percent were athetoid.

Denoff and Robinautt (1960) state that "various authors agree that over 50 percent of the cerebral palsied have oculomotor defects and 25 percent or more have subnormal vision."

Gordon (1975) reports that 25 percent of the hemiplegic cerebral palsied have a loss of vision in either the left half or right half of the visual fields of both eyes. Visual field deficits usually correlate highly with the presence of a sensory impaired limb. Strobismus and turning of the eyes inward or outward occur in approximately 30-35 percent of the cerebral population. Nystagmus (jerky eye movement) is common among ataxics (Kirk, 1972). The incidence of refractory type errors is two times as common among spastics than athegoids. Athetoids are more prone to have hyperopia; spastics may have either myopia or hyperopia (Gordon, 1975).

Perceptual disorders. One of the psychological disabilities commonly attributed to cerebral palsy is perceptual disorder. Over the past ten years, there has occurred an increased awareness of the visuoperceptual disorders that handicap cerebral palsied children. The difficulties are

TABLE 3-6

COMMUNICATION HANDICAPS IN VARIOUS TYPES OF CEREBRAL PALSY

Cerebral Palsy Type	Percentage of Each Type in Total Group	Percentage with Defects		
		Vision	Hearing	Speech
	N=1406	N=1297	N=1293	N=1224
Spastic	45.9	27.3	7.2	52.0
Athetoid	23.7	20.4	22.6	88.7
Rigidity	12.6	29.0	13.7	72.2
Ataxic	10.8	42.7	18.4	85.3
ALL GROUPS		27.6	13.3	68.0

Source: Hopkins, T. W., Bice, H. W., and Colton, K. C. Evaluation and Education of the Cerebral Palsied Child. Washington, D.C. Council for Exceptional Children, 1954.

TABLE 3-7

LIFE FUNCTION TABLE - CEREBRAL PALSY: COMMUNICATION

Statement of Problem	Dynamics	Source
Visual anomalies of varying degrees due to eye muscle abnormalities.	Ataxics show greatest number of visual abnormalities.	Kirk, 1972; Scherzer, 1974; Hopkins, et al. 1954; Denorr & Robinault, 1960.
Visual field deficits.	25% of hemiplegics.	Gordon, 1975.
Strobismus, turning of eyes inward, outward.	30-35% of cerebral palsied population.	Gordon, 1975; Abbot, 1957.
Nystagmus or jerky eye movements.	Most common among ataxics.	Kirk, 1972; Abbot, 1957.
Refractory type errors.	Most common among spastics.	Gordon, 1975.
Hyperopia	Especially among athetoids.	Gordon, 1975.
Myopia	Athetoids, spastics.	Gordon, 1975.
Difficulties in recognizing shapes and patterns; copying and reproducing forms.	Especially in spastics.	Koupernik, et al., 1975; Molnar & Taft, 1973; Cruickshank, et al., 1957.
Spatial inability: difficulties in distinguishing distances or nearness of objects.	Especially among younger children.	Koupernick, et al., 1975; Densdon, 1952.
Deficient capacity to perform auditory-visual integration.		Schain, 1972.
Various hearing defects - i.e., patient hears the high frequency sounds, but misses the low.	Especially among athetoids.	Fish, 1955; Bakwin & Bakwin, 1972; Hopkins, et al., 1954; House, et al., 1972.
Muteness.	Occurs in some.	Bakwin & Bakwin, 1972.
Language disabilities due to multiple impairments: perceptual disturbances, hearing loss, neuromuscular abnormalities.		Hull & Hull, 1972; Myers, 1965.
Speech defects in approximately 70% of cerebral palsy patients: (1) difficulty in coordinating the speech mechanism muscles to produce even the simplest sounds.		Denoff & Robinault, 1960; Hagen, et al., 1973; Kirk, 1972.

TABLE 3-7, CONT'D.

Statement of the Problem	Dynamics	Source
(2) Dysarthria		Hagen, et al., 1973.
(3) Stuttering		Kirk, 1972.
(4) Aphosia		Kirk, 1972.
(5) Articulation lacks preciseness	Especially among athetoids.	Kirk, 1972.
(6) Voice may be pitched too high or too low	Especially among athetoids.	Kirk, 1972.
(7) Weak in vocalization		
(8) Speech labored, indistinct, husky, guttural.	Especially among spastics.	Kirk, 1972.
(9) Difficulties in assimilation of sounds and intonational patterns.	Especially among ataxic.	Kirk, 1972.
Tactual and kinesthetic impairment in perceiving weight, shape, and texture.		Woods, 1957.

not encountered in all cerebral palsy patients, and the severity and extent of the disorders also varies greatly (Koupernick, et. al., 1975; Mackeith & Williams, 1975).

Visuoperceptual disorders may occur in the presence of normal intellectual functioning and can seriously impair academic performance (Molnar & Taff, 1973).

Characteristics of visuoperceptual abnormalities associated with cerebral palsy include recognition of shapes and patterns; sorting or matching according to shape; copying or reproducing forms from memory (Koupernik, et. al., 1975). Cerebral palsied children often have difficulties in spatial orientation including difficulties in discriminating figure and background and recognizing position (Cruickshank, et. al., 1957).

Among young cerebral palsied children, there is often difficulty in distinguishing the nearness and distance of furniture and other objects as they move about a room. This is called "spatial inability" and is important in that it hinders normal educational progress. Spatial ability is essential in the process of learning to read and write (Koupernik, et. al., 1975).

Spastics are more prone to have perceptual problems than are children with athetoid or ataxic types of cerebral palsy, though all three types have impaired perception more often than the other types (Cruickshank, Bice & Wallen, 1957).

Cerebral palsy patients are often deficient in their capacity to perform auditory-visual integration, due to a defective capacity for intersensory integration (Schain, 1972).

Hearing. Since hearing defects do not involve motor functions comparable to those of vision or speech, one might expect hearing defects to be less frequent among the cerebral palsied. Research has found that hearing defects among cerebral palsied children are, in fact, less common than are visual problems (Kirk, 1972). Most researchers estimate incidence of hearing impairment among the cerebral palsied at around 25-35 percent (Bakwin, 1972).

Hopkins, Bice and Colton (1954) reported that 13.3 percent of 1,121 cases of cerebral palsy had hearing defects. Athetoids comprised 22.6 percent of the sample; spastics only 7.2 percent. In an English survey Fish (1955) found that 20 percent of the cerebral palsy cases he studied involved hearing losses.

Hearing defects are frequent in children with kernicterus. The hearing loss is usually greatest between 2,000 and 4,000 cycles per second with a slight improvement at around 8,000 cycles (Abbot, 1957).

Speech disorders. Defective speech is the most common type of handicap associated with cerebral palsy (Kirk, 1972). A number of researchers (Denhoff & Robinault, 1960) indicate that because of neuromuscular involvement, speech defects exist in 70 percent of cerebral palsied children, being more common in athetoids than in spastics. Many cerebral palsied children find it extremely difficult to coordinate the movements of the speech mechanism to produce even the simplest of sounds. Early verbal stimulation and "formal" training by specialists are desirable before the child begins the first grade (Hull & Hull, 1972). Some cerebral palsied children never acquire speech, though one-half to three-fourths can be benefitted by speech training (Bakwin & Bakwin, 1972).

Cerebral palsied children who are also mentally retarded and can not learn to modify aberrant patterns of neuromuscular control due to severity of the muscular involvement should be taught a non-verbal form of communication (Hull & Hull, 1973).

Speech impairments range from mild dysarthria (an articulation defect caused by poor motor control) to a complete inability to speak. Other speech defects include delayed speed due to mental retardation and the cerebral dysfunction, voice disorders, stuttering, and aphasia (Kirk, 1972).

The neuromuscular nature of speech defects is obvious. Rigidity of facial muscles may cause articulation to lack precision, and there might be substitutions or omissions of sounds. The voice may be pitched too high or too low because of involvement of larnyx (Bakwin & Bakwin, 1972). The respiratory mechanism may fail to produce coordinated breathing patterns, hindering the air flow necessary for sound production. Sometimes reversal patterns of breathing may occur, and the child may attempt to talk upon exhalation. In the presence of such respiratory involvements, speech will be weak in vocalization and aspirate in quality. Easy rhythmical responses are affected by respiratory deviations thus causing speech patterns to be scanning, spasmodic, staccato, or explosive, depending on the kind of control the patient tries to exert (Kirk, 1972).

The speech of the spastic shows greater articulatory deviations than does the speech of the other types. Speech may be labored and indistinct and husky, guttural and tense in quality. Saliva is likely to be excessive in sound formation (Kirk, 1972).

The athetoid manifests slurred speech which is constantly changing in pitch. Sounds are distorted inconsistently because of the continuous

involuntary movements characteristic of this condition. Often the voice lacks force due to respiratory disorders and the excessive movement (Kirk, 1972).

The ataxic talks in the same rhythm shown in his walk and bodily movements. The speech will exhibit spasmodic breaks and pauses rather than slurring or scanning rhythm. At times, speech may seem to fade away. The ataxic has the most difficulty in the assimilation of sounds and intonational patterns (Kirk, 1972). Speech may also be delayed or inadequate partly because parents do not allow the child opportunity to exercise his vocal musculature. Speech and language development are aided by the stimulation provided by the child's exploration and experience of the environment. The cerebral palsy child, being restricted in his motor capabilities, may require extra stimuli.

If is often difficult to distinguish between speech and language disorders in children with multiple impairments. Since brain lesions are often widespread, marked language disturbances are often present. The presence of perceptual disturbances, of the visual, auditory and tactile-motor modalities also interferes with development of language concepts. Neuromuscular involvement limiting the cerebral patient's ability to move about his environment also contributes to linguistic development. Hearing loss also affects the child's ability to discriminate between speech sounds and thus interferes with normal language acquisition (Hull & Hull in Dunn, 1972).

Myers (1962) using the Illinois Test of Pscyholinguistics, studied the language disability patterns of children with cerebral palsy. Spastics performed better than other cerebral palsied individuals on the automatic sequential items of the Illinois Test of Psycholinquistics, while athetoids were superior on the representational or meaning level of the language process.

Cognitive-Intellectual. Often the associated handicaps such as poor speech and uncontrolled or spastic movements of the cerebral palsied give the impression that the patient is mentally retarded. There is actually little direct relation between intelligence and the degree of physical impairment in cerebral palsy (Kirk, 1972). In a typical population of cerebral palsied children in the United States, 33 percent are mentally normal and essentially self-sufficent. Assimilation into the general population is possible for this group, though a few would need special vocational guidance or a sheltered environment. Five percent are mentally bright but have marked physical handicaps and need socialization and intellectual stimulation. For another 25 percent, mental retardation is a major problem, though physical handicaps are minimal in this group. These individuals need sheltered environment, and/or custodial or institutional care. The remainder (12 percent) present intermediate problems of mental or physical disabilities (O'Reilly & Elliot, 1971).

Although some cerebral palsied individuals are mentally retarded and/or physically disabled, the majority are trainable. According the O'Reilly and Elliot (1971), from a total of 1,513 patients, 60.8 percent were trainable. The term trainable refers to the ability to benefit from treatments which enable cerebral palsied individuals to become physically self-sufficient, even though some are mentally retarded. The remainder, 39.2 percent, were labelled untrainable: so physically and/or mentally disabled as to be essentially custodial problems. Table 3-8 breaks down these percentages further by grouping the cerebral palsied by type.

Because of the many handicapping conditions found among children with cerebral palsy, an accurate appraisal of their educational potential is

TABLE 3-8

EDUCATIONAL POTENTIAL OF THE CEREBRAL PALSIED

Prognosis	Trainable (%)	Untrainable (%)
Spasticity	78.1	21.9
Athetosis	54.3	45.7
Ataxia	71.2	28.8
Rigidity	0.0	100.0
Atonia	27.3	72.7
Tremor	20.0	80.0
Mixed	27.8	72.2
TOTAL	60.8	39.2

Source: O'Reilly and Elliot, The Future of the Cerebral Palsied Child. Developmental Medicine and Child Neurology, 1971, p. 636.

extremely difficult. There is some question as to whether the potential of the cerebral palsied child can be assessed with the same intelligence tests used with unimpaired children (Kirk, 1972). Haussermen (1958) and Taylor (1961) have developed procedures for adapting test items for evaluation of the potential of children with severe and multiple disabilities. They contend that these procedures give a more accurate picture of capabilities than can be obtained by standardized test administration.

Robinson (1973) in a study of 80 cerebral palsied subjects suggested two major learning deficit categories existent among the cerebral palsied--severely subnormal and educationally subnormal. The severely subnormal are characterized by the absence of skills usually acquired by children aged less than 50 percent of the subjects' age which could not be wholly explained by the effects of other handicaps also present. The educational subnormal manifested an absence of skills usually acquired by children aged between 50-80 percent of the subjects' age, which could not be wholly explained by the effects of other handicaps.

The high incidence of mental retardation, about 50 percent, among cerebral palsied children is largely due to the significant reduction of perceptual and cognitive skills (Andrews, 1972; Bakwin & Bakwin, 1972). Learning disabilities, emotional liability, short attention span or perserverance, low frustration thresholds, impulsivity, and distractibility are all often due to an uneven perceptual-cognitive profile (Gordon, 1975).

Molnar and Taft (1973) assert that intellectual impairment is the most serious associated handicap in cerebral palsy. They point out that mean IQ scores of the total cerebral palsy population are lower than that of the general population. There is a greater frequency of mental deficiency

and a lower incidence of above-average intelligence among the cerebral palsied than among the population at large. Approximately one-third of all cases of cerebral palsy fall into the normal range of IQ scores and an additional one-third are educable. The greatest proportion of higher IQ scores is found among athetoids. Severe retardation is found most often among rigid types (Molnar & Taft, 1973). Schonnell (1956) tested the IQ levels of 354 cerebral palsied patients. Twenty-four percent were found to have IQ's of 90 or over; with 4.0 percent of these having IQ's 110 and over (See Table 3-9 and 3-10).

Severity of the neuromotor deficit and presence of seizures have been significantly correlated with severity and frequency of mental deficits in cerebral palsy patients (Molnar & Taft, 1973). Athetoids are usually of higher intelligence and have a much lower incidence of convulsive disorders than do spastics. Children with the rigid types of cerebral palsy are usually severely retarded and have a high incidence of convulsions (Gordon, 1975).

Any child who has poor sensory feedback mechanisms, either through peripheral impairment, touch, proprioceptive or high cortical defects which impair kinesthesia will have an intellectual deficit (Gellhorm, 1949). There are different degrees of impairment depending upon the degree of disability. Learning ability is dependent upon sensory feedback; that is, if the child has some deficient sensory feedback and has been placed in a non-functional wheelchair, he has no opportunity to explore his environment. Poor postural attitudes inhibit the ability to develop a vertical visual relationship. The child will have no sense of space, texture of depth, which are the building block of learning (Jones, 1960; Gellhorm, 1950). See Table 3-11.

TABLE 3-9

IQ DISTRIBUTION AMONG NORMALS AND CEREBRAL PALSIED PATIENTS

IQ Level	% in Cerebral Palsied Group	% in Normal Population
130+	.6	3
110-129	3.4	24
90-109	20.1	46
70-89	26.8	24
69 and below	45.2	3
Not assessable	3.9	

Subjects were 354 cerebral palsied patients in England

Source: Schonnell, F.E. Educating Spastic Children, Edinburgh: Oliver and Body, 1956.

TABLE 3-10

COGNITIVE-INTELLECTUAL HANDICAPS IN VARIOUS TYPES OF CEREBRAL PALSY

Cerebral Palsy Type	Percentage of Each Type in Total Group	Percentage with Defects	
		IQ Below 70	Lack of Hand Dominance
	N=1406	N=933	N=1224
Spastic	45.9	44.1	19.1
Athetoid	23.7	40.9	36.1
Rigidity	12.6	66.3	18.0
Ataxic	10.8	70.5	32.9
ALL GROUPS		49.0	24.7

Source: Hopkins, T. W., Bice, H. V. and Colton, K. C. Evaluation and Education of the Cerebral Palsied Child. Washington, D.C., Council for Exceptional Children, 1954.

TABLE 3-11

LIFE FUNCTION TABLE - CEREBRAL PALSY: COGNITIVE-INTELLECTUAL

Statement of Problem	Dynamics	Source
Mental retardation.	In approximately 50% of cerebral palsied children.	Andrews, 1972; Bakwin & Bakwin, 1972.
Problems in conceptualization and associations: Concrete rather than abstract responses.		Dolphin & Cruickshank, 1951.
Hyperactivity, short-attention span: Often due to uneven cognitive-perceptual profile.		Gordon, 1975.
Severe retardation.	Rigid type, especially.	Molnar & Taft, 1973.
Lack of sensory feedback impairs learning, reduces perceptual skills.		Gellhorn, 1950; Jones, 1960; Andrews, 1972; Bakwin & Bakwin, 1972.

In a 1954 study of cerebral palsied children, Hopkins, Bice, and Bolton reported that 25 percent lacked established hand dominance. Many studies have indicated that hand dominance plays an important role in learning to read, although there is considerable disagreement on this point (Harris, 1970). See Table 10.

Social Attitudinal. There is no evidence to indicate that a handicapped person is automatically maladjusted. General agreement is that there is no substantial evidence that groups with impaired physiques differ from the unimpaired in general or overall adjustment or that types of physical disability related to particular personality and adjustment characteristics (Wright, 1960). However, some researchers have directed their attention to the particular personality and adjustment problems of the cerebral palsied. Allen and Jefferson (1962) ascribe to the cerebral palsied such personality difficulties as lack of recognition of the existing handicap, unconscious rejection of the self, unresolved dependency feelings, egocentricity with expansive self-concept, hostility and compensation for feelings of inferiority and inadequacy. Negative attitudes towards self and others are understandable in thatthe cerebral palsied individual is denied normal group experience due to physical limitations and sometimes because of family prohibitions (Allen & Jefferson, 1962).

Many cerebral palsied individuals are judged to be mentally ill or mentally retarded when, in fact, they may be intellectually normal. Athetoids, though usually mentally normal, display excessive involuntary motion, which involves facial and speech muscles. Grimacing and contraction of the facial muscles may give the impression that the individual is mentally retarded (Abbot, 1957). Children suffering from the tremor type may have

spasma of laughter or great crying spells with no underlying emotional feelings. They are often thought, by the layman, to be mentally and emotionally unstable (Abbot, 1957).

Moed and Litwin (1966) found that psychosocial adjustment of the cerebral palsied individual was related to: extent to which the person exaggerated his degree of ability; interest in and ability ot maintain relations with other handicapped persons; ability of the parents to permit social independence commensurate with physical capabilities; parental understanding that independence is not synonymous with self-sufficiency; parents' own attitudes and adjustment; degree of isolation because of inability to travel resulting from physical and social restrictions; and early emphasis on total rehabilitation rather than medical therapy alone.

Particular problems of adjustment are found in adolescent cerebral palsied. At this time, due to physical and psychogenic factors, the patient may appear to "degenerate". This refers to a progression of an orthopedic deformity or a spurt in bone growth. This, accompanied by insufficient muscular development, may cause the loss of a particular skill, such as walking (Gordon, 1975). See Table 3-12.

VI. Functioning as a Member of Society

The literature provides information about functioning in the labor force and in the community.

Functioning as a Member of the Labor Force

Cerebral palsied individuals seem to have the greatest difficulty in finding employment of all disabled groups. The problem stems from both employer reluctance and severity of impairment (Wilson, 1973).

The President's Committee on Employment of the Handicapped (1967) reported 200,000 cerebral palsied individuals to be of working age. Out of this 200,000 only a handful were actually employed.

TABLE 3-12

LIFE FUNCTION TABLE - CEREBRAL PALSY: SOCIAL-ATTITUDINAL

Statement of Problem	Dynamics	Source
Negative attitudes toward self and others, especially when motor disabilities are severe.	Applies to all types of cerebral palsy.	Allen & Jefferson, 1962.
Feelings of isolation.	Adolescent cerebral palsy patients.	Gordon, 1975.
Depression, despondency due to "degenerative" effect of orthopedic deformities.	Adolescent cerebral palsy patients.	Gordon, 1975.
Social withdrawal.	Applies to all types of cerebral palsy.	Hull & Hull, 1973.

Moed and Litwin (1963) reported on two studies to determine the employability of persons with cerebral palsy. The first study was conducted with ambulatory individuals over a five-year period for the purpose of establishing practical methods of work evaluation. Factors found to be most related to employability were: verbal IQ, handwriting, vocational adjustment, severity of gait defect, ability to travel independently, performance, IQ and speech intelligibility. Of those subjects who were employed at the time of the study approximately half held unskilled jobs and one-fourth were clerical workers. The clerical workers tended to stay on the job longer and were the more stable employees. Those who were employed immediately after finishing high school were more stable than those who went to work after a period of idleness. Employment chances appeared best of the first job was found within eight months of completion of high school (Moed & Litwin, 1963).

Moed and Litwin's second study evaluated young cerebral palsied adults from vocational centers in the United States. They concluded that over 50 percent of the persons they studied needed sheltered workshop employment and estimated that 60-70 percent of the cerebral population are marginal workers without the capacity for regular employment (Moed & Litwin, 1963).

Machek, Hardin and Collins (1960) described a project for comprehensive evaluation and classification of the vocational potential of individuals with cerebral palsy. Slightly less than one-half of the individuals screened were judged to have vocational potential. For the severely impaired, vocational potential was found to be poor. Only one-fourth could be placed on jobs, and the great majority appeared to be able to be productive only in a sheltered workshop, since the tensions of regular employment tended to increase motor problems and multiply emotional defects.

Cerebral palsied individuals from smaller communities and those having less agency exposure were inclined to do better work and to adjust more readily. The authors concluded that the most important factors in predicting vocational adjustment and success were not IQ or sample job performance but motivation and emotional stability. Many of the young people in the study were found to have a low level of maturity, an unrealistic attitude toward employment and lower chances for vocational rehabilitation. Results stressed the placement of emphasis on the total adequacy of the individual, including qualities that pertain to the practical application of intelligence (Mackek, Hardin, & Collins, 1960).

Functioning as a Member of the Community

Cerebral palsy places great strain on the person with the disorder and his family. In the beginning, most parents fight fiercely against the facts, and many parents do not altogether accept their handicapped child. Often they refuse to accept the extent of the child's disability or respond by overprotecting the child so that he cannot grow. Often professional counseling is needed to help both families and people with cerebral palsy face the situation (Denoff, 1966).

The cerebral palsied adolescent faces new tensions and new needs. For one thing, he is now bigger and heavier and much harder to handle physically, if that is necessary. Also, people inevitably expect more of a teenager than of a baby; he is less appealing in his helplessness. Yet the cerebral palsied adolsecent, like every other, needs to try to outgrow his dependence on the family (so much greater in his case). He needs tolerance for his rebellions, guidance for his uncertainties and security in family relationships

to support him during this crisis period. It becomes even more distressing when he is misjudged or mislabeled by other as mentally retarded or emotionally disturbed (Abbott, 1957).

Speech defects may cause the cerebral palsied adolescent to withdraw from society (especially his peer group) and/or to become hostile toward the rest of society. The speech of the cerebral palsied may not be socially acceptable once a certain state of growth has been surpassed (Hull & Hull, 1973). Drooling and a protruding tongue may also cause the cerebral palsied individual to be the object of social rejection.

To help families meet and solve their problems, cooperative efforts are now being made in many locations. Local citizen groups, government agencies for health, medical centers, organizations for social welfare and other special services, and private health agencies are working together (See Service Delivery section).

VII. Technologies

Since cerebral palsy is noncurable, sufferers must learn to live with the disability from infancy. Treatment technologies are aimed primarily at relieving the symptoms of the various types of cerebral palsy. The efficiency of treatment is dependent on many factors, not the least of which is early detection. The treatment process circumvents the areas which are damaged and utilizes the unimpaired areas to help perform the necessary functions. A child must learn to relax those muscles which become tense and prevent motion, to synchronize muscle groups for smoothness of movements, and to bring uncontrolled movement under voluntary control (Wilson, 1972).

Procedures advocated by various groups differ to some extent. There is general agreement on the proximal-distal chronology in treatment and also on proceeding on a gross to fine continuum. There is considerable disagreement on the use of bracing, the decision to use or resist "natural" lesion-produced peculiarities of motion, the importance and function of residual reflex activity and the significance of sensory feedback. Disagreements seem to be related more to a question of emphasis than to the exclusive use of particular methods of therapy (Weiss & Betts, 1972).

More recently there has been a trend toward a "global" treatment approach. This approach considers the child, the family, and the community in a total effort towards rehabilitation (Cruickshank & Rauss, 1955). Emphasis is placed on the child's assets and strengths; it is believed that assessment of the patient's strengths and assets is as important as evaluation of deficits. An effort is made to capitalize on sensory, muscle, and intellectual assets to offset deficits, and achieve function. The chief goal of this type of treatment is the achievement of efficient function; cosmesis is secondary. An active follow-up is necessary to secure the child's continuing integration into both the family and the community. New problems may arise and new abilities emerge with growth, and each must be taken into consideration (Weiss & Betts, 1972).

Method of Treatment

1. Preventative technologies, public education programs concerning causes of cerebral palsy (Molnar & Taft, 1973).
2. Aims of treatment (Bobarth & Bobarth, 1967).
3. "Sensory Story" technique to increase use and awareness of affected limbs in hemiplegic children (Barret & Jones).

4. Comparison of classic and "sensory-motor learning" techniques (Bobarth & Bobarth, 1967; Bleck & Handley).

5. Surgical techniques to prevent deformity and prevent occurrence of deformity (Wilson, 1973; McCarroll, 1972; Denoff, 1966).

6. Use of splints and braces in controlling deformity (McCaroll, 1972; Wilson, 1973; Phelps, 1963).

7. Importance of early diagnosis and treatment in rehabilitation technologies (Bobarth & Bobarth, 1967; Andrews, 1972; Weiss & Betts, 1972).

8. Physical equipment and facilities needed in schools for the cerebral palsied (Wilson, 1972).

9. Importance of early treatment for speech defects (Hull & Hull, 1972).

10. Operation on the brain to relieve symptoms (Masland, 1967; Denoff, 1972).

11. Electric stimulation of the brain to create theurapeutic lesions (Wilson, 1972).

12. Use of special exercise equipment and programs (Phelps, 1963; Fay, 1954; Doman, 1960).

13. Pathological unlocking reflexes and rehabilitation of spastics (Fay, 1954; Doman, 1960).

14. Learning of body postures to improve functional abilities (Bobarth & Bobarth, 1952).

15. Proprioceptive neuromuscular facilitation technique (Kabat & Knott, 1953; Bellhorn, 1949, 1950; Rood, 1956).

16. Sensorimotor interfacilitation technique (Rood, 1956; Deaver).

17. Capitalization method (Weiss & Betts, 1972).

18. Use of drugs in treatment (Denoff, 1972).

19. Multiple-sense modality approach in speech therapy (Kirk, 1972).

20. Total or global treatment philosophy (Paine, 1962; Cruickshank & Rauss, 1955; Molnar & Taft, 1973; Weiss & Betts, 1972).

21. Treatment modalities in various combination (Molnar & Taft, 1973).

22. Language boards; picture, word and phrase boards (MacDonald & Schulz).

23. Non-verbal electronically mediated communication (Lavoy, 1957; Jones, 1961; Maling & Clarkson, 1963; Hill, et.al., 1968; Larson, 1971; Reallock, 1958; Hagen, et. al.).

24. Behavior-shaping techniques (Stone, 1970; Fassler, 1969).

25. Psychiatric practices with the cerebral palsied (Allen & Jefferson, 1962).

VII. Service Delivery

Traditionally, cerebral palsied children have been placed in special schools and classes. The trend is now away from such specialized facilities except for the severely and multiply handicapped. The cerebral palsied have also been placed in residential schools for thorough diagnosis and evaluation and sometimes as permanent residents. Often, such placement is based upon the presence of associated disabilities such as visual or learning disabilities. Often such schools are equipped to deal only w[illegible]ich disabilities and not the overall condition of the child. Recently, however, many residential schools are recognizing their responsibility and are providing the special facilities and trained personnel required.

Increasing numbers of cerebral palsied children are being moved into special day schools and classes closer to their homes where they can receive their education in a less segregated situation (Wilson, 1973).

In some areas, in an effort to develop appropriate community attitudes, community auxiliaries have been organized. The purpose of these groups is to follow-up on the cerebral palsied child, and become more acquainted with the needs of the cerebral palsied. They organize architectural projects to secure removal of barriers. Often, educational programs are held to improve community attitudes (Weiss & Betts, 1972).

A number of groups have become involved in aiding the cerebral palsied population. United Cerebral Palsy Associations, Inc. (incorporated in 1949) is a federation of more than 300 self-governing state and local groups. This organization has as its goal an overall, long-range attack on

the entire problem of cerebral palsy. The organization provides treatment, care, and education for the cerebral palsied population; and trains them for employment as well as finding jobs for them. The United Cerebral Palsy Research and Education Foundation, established in 1955, finances research grants and professional training opportunities for those involved in cerebral palsy studies.

Certain national groups have contributed to programs of care and to increased understanding of cerebral palsy. These groups include: the National Society for Crippled Children and Adults, the American Public Health Association, the American Academy for Cerebral Palsy, and various agencies of the United States Department of HEW (Children's Bureau, National Institutes of Health, Office of Vocational Rehabilitation).

Research in cerebral palsy is being sponsored by the National Institute of Neurological Diseases and Blindness of the National Institutes of Health, the United Cerebral Palsy Research and Education Foundation, the Easter Seal Research Foundation, and the Association for the Aid of Crippled Children.

The United Cerebral Palsy Association, Inc., a voluntary health agency:

1. Promotes research.
2. Promotes professional and public education.
3. Promotes more adequate techniques and facilities for diagnosis and treatment
4. Promotes employment of cerebral palsied.
5. Raises funds.
6. Establishes and works with local and state facilities.

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